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DEPARTMENT
OF AGRICULTURE

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Eperythrozoonosis in Sheep.

By W. O. NEITZ, Section of Protozoology and Virus Diseases, Onderstepoort.

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1. INTRODUCTION.

THE presence of *Eperythrozoon ovis* in the blood of sheep at Onderstepoort was reported by Neitz, Alexander and du Toit in an address to the Biological Society of Pretoria, on March 15th, 1934, and was recorded in this journal the same year. Since the appearance of that original publication research into the problem of Eperythrozoonosis has progressed. The results obtained form the subject of this paper.

It will be seen, however, that there remains a considerable amount of work to be completed, but it is considered that the publication of an interim report is justified owing to the slow progress being made, chiefly due to the fact that 15-20 per cent. of available experimental sheep harbour a latent infection of the parasite.

Frequent references were made to the work on Oroya fever in man and *Bartonella* and *Eperythrozoon* infections in rats and mice since those studies have proved an invaluable guide to investigations into the analogous disease in sheep.

2. DEFINITION.

Eperythrozoonosis of sheep is an infectious disease caused by *Eperythrozoon ovis*, a small supra- and inter-cellular blood parasite having a ring, rod, irregular or oval shape and belonging to the family *Anaplasmidae*. Initial invasion of the organism results in the production of irregular pyrexia, a variable degree of icterus, and anaemia characterized by the appearance of degenerative and regenerative elements in stained blood films. Relapses at varying intervals after recovery may occur. The mode of transmission is not known, but it would appear that the vector is a blood-sucking arthropod.

3. HISTORY.

It is difficult to ascertain to what extent unidentified infection of *Eperythrozoon ovis* has been a complicating factor in previous work on anaplasmosis, heartwater, bluetongue, trypanosomiasis, verminosis and other diseases in sheep. For instance, de Kock and Quinlan (1926) in their report on the results of splenectomy in sheep state " Another observation to be recorded in connection with the erythrocytes of some of the sheep is the appearance of a peculiar irregular reticular-like network. These varied in shape from long irregular threads to network-like masses resembling large piroplasms. No chromatin or typical cytoplasm could be identified in them ". In some sheep they observed these structures alone, in others they were associated with *Anaplasma ovis*, but the nature of these irregular reticular masses remained unexplained. It would appear now that an infection with Eperythrozoon had been encountered. Similar structures have been observed by other workers when examining stained blood films, but in each case they were dismissed as extraneous material, stain deposit or artefacts. Since the recognition and description of the parasite by Neitz, Alexander and du Toit in 1934, it has become apparent that certain unexplained anaemic changes in the absence of anaplasmosis or verminosis can now be accounted for.

Nothing is known of the rôle played by this parasite in the field. Farmers in South Africa describe an anaemic condition in sheep to which the name bleeksiekte (pallor disease) has been given, but the aetiology is quite obscure and it is not known to what extent verminosis and malnutrition in addition to *Ep. ovis* are contributory factors.

4. OCCURRENCE.

Little is known of the distribution of this condition in South Africa since it has not been possible to carry out a methodical survey. The majority of the Merino sheep used for experimental purposes at Onderstepoort are purchased in the Karroo, chiefly in the vicinity of Middelburg and De Aar in the Cape Province. Examination of these animals has revealed the presence of individuals showing active as well as latent infections. Further, the presence of infection has been diagnosed in Blackhead Persian sheep introduced into the Pretoria district from the Northern Transvaal. Bearing these observations in mind, together with the normal exchange of sheep at sales, the widespread practice of moving flocks of sheep during the winter months from the highveld to the lowveld, and the necessity of trekking in search of grazing during periods of prolonged drought, the belief is justified that *Ep. ovis* is widely disseminated throughout South Africa.

The occurrence of *Ep. ovis* in sheep has been described by Donatieu and Lestoquard (1935) in Algeria, by Delpy (1936) in Iran and by Lafenetre (1936) in France.

5. AETIOLOGY.

(a) *Classification.*

The close resemblance between *Eperythrozoon* on the one hand and *Bartonella* and *Grahamella* on the other hand, together with the apparent relationship to *Anaplasma* has been mentioned by Kikuth and other investigators. Neitz, Alexander and du Toit (1934) suggested that these four genera should be included in the family *Anaplasmidae*, but the exact position of the parasite in any scheme of classification has not been decided upon finally.

The description given is that of the appearance of the parasite seen on examination of rapidly dried blood films fixed by May-Grunewald and stained with Giemsa. Stained by this technique the organisms take on a delicate pale purple to pinkish purple colour. Typically they are seen as delicate rings approximately 0·5-1 μ in diameter though occasionally they may be somewhat larger. In addition to ring forms it is common to encounter triangles with rounded angles, ovoid, comma, rod, dumb-bell and tennis racket forms.

It has been observed frequently that at one end of the smear ring forms predominate while towards the other end the number of rod and comma forms is in the majority. It is believed that this distribution is purely mechanical, being brought about during the process of drawing the blood film.

(c) *Localization.*

Although large numbers of organisms are to be found lying supra-cellularly on the erythrocytes in blood smears, the majority appear to be free between the cellular elements, but this distribution varies within very wide limits. Usually the number of supra-cellular forms is directly proportional to the intensity of the infection, and it has been observed frequently that during the first 24 or 48 hours after the appearance of the parasites practically all the organisms are to be found lying on the erythrocytes; as the disease progresses the proportion of free forms increases.

(*a*) *Supra-cellular Forms.*—A single organism may be present, but on the other hand the entire surface of a red cell literally may be covered with parasites which seem to be lying one on top of the other. Most commonly the supra-cellular forms are to be found in clusters of 3-12 aggregated towards the centre of the cell, or at a point towards the periphery or actually along portion of the border. A fairly characteristic picture is to find several rod shapes strung together along the periphery of a cell in such a way as to form a partial or complete ring. In some cases a very fine fibre commencing from a cluster or circle of ring forms, may be seen drawn a variable distance across the cell like a veil.

(*b*) *The Extra-cellular or Free Forms.*—These usually predominate and are fairly evenly distributed throughout the preparation. It has been observed sometimes in thick smears where the erythrocytes are packed together that the interstices are filled with a homogeneous mass that stains in a manner similar to the parasites.

It is not known whether the distribution of *Ep. ovis* in stained smears is a true picture of the distribution of the parasites in the circulation. After centrifuging defibrinated or oxalated blood for 1 hour at 3,000 revolutions per minute the majority of Eperythrozoa could be demonstrated in the layer just below the leucocytes. This indicates that the union between the parasites and the host cells is rather loose so that it is possible that a large number of the extra-cellular organisms may simply have been detached during the process of smear preparation. This point of view is supported by the repeated observation that the proportion of extra-cellular to supra-cellular parasites varies considerably in different preparations made from the same animal at the same time.

It is possible that multiplication may take place by budding, since occasionally smaller forms may be seen lying in contact with those normal in size. In some of the ring forms there may be noticed 1, 2 or 3 points which stain in an appreciably darker colour. The significance of these points is quite obscure, but they may stand in some relation to multiplication.

All attempts to cultivate *Ep. ovis* on the usual laboratory media have been unsuccessful.

6. TRANSMISSION.

Nothing is known about the natural mode of transmission of *Ep. ovis* in sheep. In the *Eperythrozoon* infection of mice the louse (*Polyplax serrata*) has been found to be the vector. No ectoparasites were found on a few Merino sheep that were showing an active infection of *Ep. ovis* which had been contracted naturally.

The intravenous injection of emulsified keds (*Melophagus ovinus*) collected from known carriers failed to set up the disease in a susceptible splenectomized sheep.

Artificially infection may be transmitted from sheep to sheep by the subcutaneous or intravenous subinoculation of blood or emulsified organs.

7. PATHOGENICITY.

Up to the present the disease has been studied at Onderstepoort only in Merino sheep, so that nothing can be said of the relative susceptibility of, and the course of the disease in, the different breeds of sheep.

The susceptibility of different adult Merino sheep varies considerably as evidenced by variations in the degree of anaemia and icterus in different individuals after artificial infection. From the limited number of cases studied it would appear that there is no difference between the susceptibility of lambs and young sheep and that of adults.

Artificial infection of a susceptible splenectomized calf was successful, resulting in the production of severe anaemia and icterus. On the other hand, a splenectomized dog, together with rabbits and guinea-pigs, proved to be refractory.

8. PATHOGENESIS.

The Eperythrozoa parasitize the erythrocytes, but it is not clear how destruction is brought about. Varying with the intensity of infection a larger or smaller number of red cells is destroyed resulting in the production of anaemia. Examination of the blood has shown that there is a rapid drop in the red precipitate obtained on centrifugation and the red cell count may fall as low as 1,000,000 per c.c. within 10 days. The decrease in the number of erythrocytes commences at the time of the first appearance of parasites. Simultaneously there is a rise in the leucocytic count up to 20,000 per c.c. This increase is accounted for by an absolute and a relative monocytosis. Erythrophagocytosis is well marked. With the development of anaemia evidences of degenerative and regenerative processes are seen in stained smears, namely anisocytosis, polychromasia, punctate basophilia, reticulocytes, jolly bodies, nuclear rests and normoblasts. Haemoglobinurea has been observed in one case where the destruction of red cells proceeded with great rapidity. Icterus is generally present.

According to Graf (1935), who examined the blood changes associated with *Ep. ovis* infection from a chemical point of view, "Severe anaemia was noted, the haemoglobin decrease being up to 60 per cent. of the initial haemoglobin value, decreasing over a period of three weeks post-infection to the minimum. Regeneration is relatively slow; three weeks after reaching the minimum level only approximately 60 per cent. of the initial haemoglobin count is attained. Although the maximum erythrolysis takes place during the hyperrhexia visible haemoglobin-anaemia was never observed; no spectroscopic examination of the serum for haemoglobin was undertaken. Bilirubinaemia of the indirect van den Bergh type was present, generally also icterus. The red cell count shows an enormous decrease, from about 10×10^6 to 2×10^5—i.e. more striking than the haemoglobin decline, pointing to a high colour: count ratio; The morphological blood changes are well marked. The protein of the whole blood falls to 10 per cent. of normal, whereas the non-protein-nitrogen fraction rises slightly during the acme of the reaction, chiefly due to an increased urea-nitrogen. The amino-acid-nitrogen, the uric acid nitrogen, the rest nitrogen and total creatinine nitrogen show no significant changes ".

9. SYMPTOMS.

The symptoms and course of the disease have been studied in Merino sheep maintained under stable conditions and in a small camp where the animals were exposed to adverse climatic conditions, but where they had easy access to food and water. The observations are detailed in tabular form in Table I. The reactions in the stabled sheep did not differ from those in the sheep maintained outside in the camp except that in the latter the temperatures were usually appreciably higher and the daily fluctuations were wider. No opinion can be expressed as to the nature and course of the disease in sheep maintained under South African farming conditions where adverse climatic conditions, periodic shortages of food and water and inter-current infections such as verminosis might be complicating factors. In addition observations on a few splenectomized sheep were recorded; these are tabulated in Table II. In every instance the disease was set up by artificial means.

The average period of incubation is 5-7 days. Sub-inoculation of blood from a sheep during that period of a primary reaction when the number of circulating parasites is most numerous tends to decrease the period of incubation while a corresponding lengthening is observed after sub-inoculation of an equal dose of blood from a premune sheep during the period of latent infection.

The first appearance of parasites has been observed as early as the second day, but may be delayed as long as twenty-six days after infection by either the subcutaneous or intravenous route. On an average they are first observed on approximately the fifth to seventh day. The parasites multiply rapidly and within a week may be 25 to 100 times as numerous as the erythrocytes. They are present in greatest number usually between the fifth and tenth day after their first appearance, but this time may vary from the third to the fourteenth day. The organisms may be demonstrated microscopically in the peripheral blood for a period of 6-42 days with an average period of fourteen days. It would appear that active multiplication continues up to the time when the first signs of anaemia make their appearance in the smears. Then the number suddenly decreases so that when the anaemia is most marked comparatively few or no organisms may be seen. When the condition of the blood tends to return to normal there may be a recrudescence of infection. In those cases where parasites could be demonstrated continuously for 35-40 days a graphic representation of the daily number would show the presence of several peak periods, i.e. there was a marked fluctuation in the number of parasites circulating. Disappearance of the parasites may be followed by reappearance after an interval of days, weeks or months during which they cannot be demonstrated microscopically. In one instance (sheep 37175) careful examination of blood smears over a period of 547 days showed that subsequent to the primary reaction three relapses occurred after intervals of 11, 23 and 109 days during which the blood was free from parasites.

In the course of the disease usually there is a distinct febrile reaction. The incidence of fever may be the first symptom but it may develop only subsequent to the appearance of parasites in the blood. The temperature may rise as high as 107° F. but usually it does not exceed 105°. Fever may be continuous for three or four days or it may be intermittent. Febrile exacerbations and remissions at intervals of a week or more are common, but alternatively there may be a complete absence of hyperthermia. Irregular hyperrhexia has been observed during the period when relapses occur.

Anaemia is a characteristic and constant symptom. It may be demonstrated clinically about five to eight days after the first appearance of parasites, and may last for a month or more. As the condition progresses the visible mucous membranes become more and more pale until eventually they take on the appearance of white porcelain.

Clinical icterus persisting for a few days has been observed in several cases. It is of interest to note that although an icteric condition of the mucous membranes may not be apparent yet the serum from such animals shows a varying degree of discoloration which may be demonstrated for a week or more. Only when the serum is a dark yellow colour does jaundice become apparent in a clinical examination of the living animal.

For the rest the symptoms are those associated with fever and anaemia, namely dullness, inappetence, loss of condition and debility, rapid weak pulse and accelerated panting respirations. One case showing haemoglobinuria has been encountered.

In splenectomized sheep the course of the disease did not differ appreciably from that described in non-splenectomized sheep above. Possibly the period of incubation was somewhat shorter and the rate of multiplication of the parasites rather more rapid, but this was hardly significant.

10. PROGNOSIS.

Up to the present time mortality has been recorded once in the experimentally infected Merino sheep. No opinion can be expressed as to the possible termination after natural infection under adverse conditions in the field. Even though it would appear that mortality need not be feared, the disease if widespread must be of considerable economic importance because of the severe constitutional disturbance, the anaemia, debility and rapid loss of condition.

11. PATHOLOGICAL ANATOMICAL CHANGES.

Since mortality only occurred once during the course of these experiments post-mortem examinations were carried out on sheep destroyed at various stages during the reaction. In general it may be stated that the lesions closely resemble those seen in anaplasmosis.

TABLE 1.

Observations in Artificially Infected Non-splenectomized Sheep.

Primary Reaction.							Relapses during period of observation.				
D.O.B. No. of Sheep.	Infected from.	Total period of observation in days.	Incubation period in days	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.	No. of relapses.	Interval in days of parasite free period.	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.
35448	39466	63	2	15	6+	Severe Anaemia.	—	—	—	—	—
35449	39466	63	2	12	6+	" "	—	—	—	—	—
40937	41030	18	4	6	6+	Severe Anaemia.	—	—	—	—	—
41053	41030	19	5	9	5+	Severe Anaemia.	—	—	—	—	—
41016	41199	83	5	9	5+	" "	2	18 7	1 15	2+ 4+	Anaemia. "
41038	41424	34	5	11	5+	" "	—	—	—	—	—
41585	41968	51	5	11	6+	" "	—	—	—	—	—
41537	40968	51	5	13	5+	" "	—	—	—	—	—
41581	40968	51	5	13	6+	" "	—	—	—	—	—
41422	40951	25	5	13	5+	" "	—	—	—	—	—
41097	41199	72	5	14	5+	" "	—	—	—	—	—
41555	40968	51	5	15	5+	" "	—	—	—	—	—
37978	37397	51	5	39	5+	" "	—	—	—	—	—
37309	37385	86	5	40	5+	" "	—	—	—	—	—
41510	40968	51	7	7	4+	Severe Anaemia.	—	—	—	—	—
41530	40968	51	7	10	5+	" "	1	12	19	5+	Slight Anaemia.
36994	35798	56	7	11	6+	" "	2	7 4	15 4	5+ 3+	Anaemia.
41541	40968	51	7	19	5+	" "	—	—	—	—	—
37385	32730	105	7	20	5+	" "	—	—	—	—	—
41527	40968	51	7	23	4+	" "	—	—	—	—	—
41572	40968	51	7	26	5+	" "	—	—	—	—	—
37447	32730	52	7	28	6+	" "	2	15 16	2 1	2+ 2+	Slight Anaemia. " "
37397	32730	105	7	36	6+	" "	—	—	—	—	—
40127	39466	42	7	10	5+	" "	—	—	—	—	—

TABLE I—(*continued*).

		PRIMARY REACTION.					RELAPSES DURING PERIOD OF OBSERVATION.				
D.O.B. No. of Sheep.	Infected from.	Total period of observation in days.	Incubation period in days	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.	No. of relapses.	Interval in days of parasite free period.	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.
41532	40968	51	8	8	5+	.. ..	1	22	2	2+	—
41520	40968	51	8	11	4+	.. ..	—	—	—	—	—
41523	40968	51	8	11	5+	.. ..	—	—	—	—	—
41563	40968	27	8	12	5+	.. ..	—	—	—	—	—
35798	37874	48	8	13	5+	.. ..	—	—	—	—	—
41588	40968	51	8	38	5+	.. ..	—	—	—		—
37096	35096	131	9	11	6+	Severe Anaemia.	1	7	18	5+	—
41424	40851	26	9	7	5+	.. ..	—	—	—	—	—
41513	40968	51	9	10	4+	.. ..	2	23	1	+	—
								7	1	+	
41574	40968	51	10	8	3+	Severe Anaemia.	—	—	—	—	—
40951	41422	43	12	22	6+	.. ..	—	—	—	—	—
41001	41424	34	13	14	5+	.. ..	—	—	—	—	—
41538	40968	51	14	17	3+	.. ..	—	—	—	—	—
41107	35449	40	15	19	5+	.. ..	—	—	—	—	—
41054	41030	66	19	13	5+	.. ..	—	—	—	—	—
37175	37429	574	19	26	6+	.. ..	3	4	19	4+	Anaemia.
								23	14	5+	..
								109	14	6+	,,
35781	35798	57	26	6	5+	.. ,,	—	—	—	—	—

NOTE.

N	indicates	negative for parasites.
2+	,,	parasites very rare.
3+	,,	parasites rare.
4+	..	parasites frequent.
5+	,,	parasites very frequent.
6+	,,	parasites extremely frequent.

TABLE II.

Observations in Artificially Infected Susceptible Splenectomized Sheep.

PRIMARY REACTION.							RELAPSES DURING PERIOD OF OBSERVATION.				
D.O.B. No. of Sheep.	Infected from	Total period of observation in days.	Incubation period in days	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.	No. of relapses.	Interval in days of parasite free period.	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.
32730	—	13	4	10	6+	Severe anaemia. Died from heartwater.	—	—	—	—	—
32704	36994	533	3	25	6+	Severe anaemia.	2	7 67	10 2	5+ 2+	Anaemia. No anaemia.
32729	Pooled blood from 8 sheep.	365	18	10	5+	Severe anaemia.	—	14	18	2+	Anaemia.

In an animal destroyed after parasites had been observed for three days in blood smears, no macroscopic lesions were visible.

In sheep destroyed later in the reaction there was observed a marked anaemia sometimes associated with a variable degree of icterus not only of the subcutaneous and subserous tissues but also of the liver, lung and kidneys. The liver showed the presence of fatty degeneration. The gall bladder usually was distended with dark green viscous bile. There may be a marked hydropericardium together with a gelatinous infiltration of the subcutaneous tissues along the ventral portion of the neck. Regularly marked tumor splenis was present, this organ on section showing a reddish brown soft but not fluid pulp and extensive hyperplasia of the Malpighian bodies. Apparently this tumor splenis persists for some considerable time after recovery, in fact it still persisted in two sheep slaughtered six weeks after recovery.

Da Rocha-Lima (1926) in illustrations of endothelial cells from cases of Oroya fever in man, indicates the presence of granular inclusions. Strong and his co-workers have expressed the opinion that at some stage of the life cycle of *Bartonella bacilliformis* the parasites multiply in the endothelial cells. In the case of *Ep. ovis* infection of sheep no intracellular inclusions were found in endothelial scrapings from the jugular veins or in impression preparations of the brain.

12. Diagnosis.

Since there is no clinical symptom which may be considered pathognomic for the disease, diagnosis is dependent upon the microscopic demonstration of parasites in blood smears. In cases of severe anaemia, irrespective of association with icterus or not, eperythrozoonosis must be taken into consideration, the diagnosis being complicated by the observation that during the acute anaemic phase of the disease parasites frequently are absent from the peripheral blood stream. This necessitates either daily examination of smears over a period of days or even weeks in the hope that a recrudescence of parasites may occur or, alternatively, subinoculation of blood into susceptible sheep which in turn must be kept under observation for lengthy periods.

Should it be determined at some future date that the condition is responsible for mortality in the field, post-mortem examination alone would hardly be of assistance in arriving at a diagnosis, since the pathological anatomical changes are common to a number of other conditions or combination of conditions more frequently encountered.

13. Differential Diagnosis.

Any condition or combination of conditions which results in the production of anaemia and icterus may lead to confusion. Thus verminosis, particularly haemonchosis and gaigeriasis, pernicious anaemia of sheep caused by a virus described in Algeria, anasplasmosis, babesiosis, trypanosomiasis, enzootic and bacterial icterus, must not be lost sight of. In all such cases a final diagnosis usually will only be reached after adequate microscopic blood examination. This necessitates directing attention to the fact that undoubtedly *Eperythrozoon* has been seen frequently by many investigators over a number of years and was not recognized as a parasitic organism. Invariably the parasites were discussed as dust, dirt, stain deposit or artefacts in the preparations; a knowledge of the morphology together with a little experience and the exercise of scrupulous care in every aspect of the preparation and staining of blood smears will obviate repetition of these errors.

14. TREATMENT.

(*a*) *General.*

The nature and symptoms of the disease are such that in the first instance attention should be paid to the feeding and general hygiene of the animals. Adequate feed of high nutritive value should be made easily accessible and the animals should be housed under conditions which will minimize the effect of adverse climatic conditions. The administration of iron and copper salts to promote blood regeneration is indicated but, for the rest, specific drug therapy is the most urgent need.

(*b*) *Specific Chemotherapy.*

From the point of view of specific chemotherapy the arsenic and antimony-arsenic compounds have proved to be of the greatest value, but the practical value of specific therapy in the absence of effective measures to prevent re-infection is not quite clear. In this respect a problem analagous to that experienced in the treatment of trypanosomiasis is encountered, namely that animals " sterilized " by the use of specific drugs subsequently become fully susceptible and are liable to re-infection in an acute form; in those cases where " sterilization " is not brought about, but where the drug has had a beneficial influence on the course of the disease, a relapse in an acute form may develop at any time.

(i) *Neosalvarsan.*—The specific action of arsenical compounds such as neosalvarsan, arsalyt and tryparsamide on *Bartonella* infection of rats has been described by Mayer, Borchardt and Kikuth (1927), high dilutions of the compounds being able to bring about disappearance of the parasites within 24 hours. Kikuth (1932) and subsequently other workers were able to show that arsenobenzols also had a direct action on *Bartonella canis* infection of dogs. Small doses of neosalvarsan resulted in a temporary disappearance of parasites whereas doses of 15 mgm. per kilo body weight produced complete sterilization. The authors state that this therapy must be regarded as a " therapia sterilisans magna " in the sense of Paul Ehrlich. Bruynoghe and Vassiliadis (1929) were prompted by the close relationship between *Bartonella* and *Eperythrozoon* to investigate the effect of neosalvarsan on the latter parasite; beneficial results were obtained immediately.

During the course of transmission experiments with *Eperythrozoon ovis* infection some of the sheep used were found to be insusceptible. This was a complicating factor of importance in the

TABLE III.

D.O.B No. of sheep	Infected from, Date; Dose 10 c.c. blood i.v.	Incubation period in days.	Before treatment parasites were present for.	Dose of neosalvarsan per Kg. body weight.	Nature of injection before treatment.	Nature of infection at different intervals after treatment.											After treatment smears were examined for.	Remarks	Interval in days between treatment and reappearance of parasites.	Parasites present for.	Remarks.
						¼ hour.	½ hour.	¾ hour.	1 hour.	1½ hour	2 hours.	3 hours	4 hours.	5 hours.	6 hours.	21 hours.					
40724	35548 3/8/34	6	5 days	5 mg.	4+		—		4+	3+	3+	3+	3+	3+	3+	N	16 days.	No anaemic changes developed	No parasites seen		—
40444	35548 3/8/34	4	7 days	5 mg.	4+	—	—	—	4	4+	4+	3+	3+	3+	2	N	16 days	Marked anaemic changes developed	No parasites seen	—	
40962	35548 3/8/34	5	6 days	5 mg.	4+			—	4+	4+	4+	4+	4	4	4+	3+	16 days	Slight anaemic changes. *Ep. ovis* demonstrated for 16 days after treatment			
40997	35449 3/8/34	4	7 days	7·5 mg.	4+	—			4+	4+	4+	4+	4+	4	4	4	16 days	Severe anaemic changes developed. *Ep. ovis* disappeared 18 hours after treatment	No parasites seen		
41001	35549 3/8/34	6	5 days	7·5 mg.	4+	—	—	—	4+	4+	4+	4+	4+	2+	2+	2	16 days	Severe anaemic changes developed. *Ep. ovis* disappeared 18 hours after treatment	No parasites seen		—
40368	39466 6/7/34	6	8 days	15 mg.	4+	—	—	—	2+	N	N	N	N	N	N	N	15 days	Anaemic changes developed	21 days	13 days	No anaemic changes were observed as result of the relapse. *Ep. ovis* frequent
39853	39466 26/7/34	6	8 days	15 mg.	4+	—		—	2+	N	N	N	N	N	N	N	15 days	Anaemic changes developed	20 days.	7 days	No anaemic changes were observed as result of the relapse. *Ep. ovis* frequent
41055	39853 28/8/34	6	4 days	30 mg.	5+	3+	N	N	N	N	N	N	N	N	N	N	50 days	No anaemic changes developed	37 days	11 days	Slight anaemic changes were observed. *Ep. ovis* frequent.
41085	39853 28/8/34	3	7 days	30 mg	5+	5+	N	N	N	N	N	N	N	N	N	N	50 days	No anaemic changes developed.	36 days	11 days	Slight anaemic changes developed as result of relapse. *Ep. ovis* frequent.
41105	37847 37855 28/8/34	4	6 days	30 mg.	5+	4+	4+	3+	3+	2+	N	N	N	N	N	N	3 days	Sheep died from heartwater			—
41544	40951 19/10/34	6	5 days	45 mg.	5+	N	N	N	N	N	N	N	N	N	N	N	45 days.	Anaemic changes developed	29 days	4 days	No anaemia developed as result of relapse. *Ep. ovis* not frequent.
41027	39466 26/7/34	7	Control	Not treated	*Ep. ovis* was very frequent and could be demonstrated for a period of 11 days. Severe degenerative and regenerative changes developed in the blood.																
35490	39466 21/7/34	2	Control	Not treated	*Ep. ovis* was very frequent and could be demonstrated for a period of 12 days. Severe degenerative and regenerative changes developed in the blood.																
35448	39466 21/7/34	2	Control	Not treated	*Ep. ovis* was very frequent and could be demonstrated for a period of 15 days. Severe degenerative and regenerative changes developed in the blood.																

TABLE IV.

D.O.B. No. of Sheep.	Injected from. Date. Dose 5 c.c. i.v.	Incubation period in days.	Before treatment parasites were present for.	Dose of Std. 386 B per Kg. body weight.	Nature of infection before treatment.	Nature of infection at different intervals after treatment.									Observations after Treatment.			Immunity Test.			
						¼ hour.	½ hour.	¾ hour.	1 hour	2 hours.	3 hours.	4 hours.	5 hours.	24 hours.	Remarks	Interval between treatment and reappearance of parasites.	Parasites present for.	Interval between treatment and immunity test.	Reappearance of parasites.	Parasites present for	Remarks
41024	41839 29/1/35	4	5 days	5 mg.	5+	5+	5+	4+	2+	+	+	+	—		Slight anaemia.	35 days. ..	7 days....	110 days .	-		No reaction.
41807	„	6	3 days	5 mg.	4+	4+	1+	2+	+		—		—	—	Slight anaemia. . ..	35 days ..	9 days ..	110 days .	17 days	7 days	Slight anaemia.
41819	„	4	5 days	5 mg.	5+	5+	5	4+	3+	—	—	—	—		Slight anaemia .. .			110 days. .			No reaction
41835	41839 29/1/35	4	5 days	10 mg.	4+	4+	+	—		—	—	—	—	—	Slight anaemia...			110 days...	17 days	29 days	Marked anaemia.
41836	„	4	5 days	10 mg.	4+	2+	—	—	—						Slight anaemia.. .	—		110 days..	17 days .	10 days.. .	Marked anaemia.
41858	41839 29/1/35	4	5 days	20 mg.	4+	2+	—	—	—	—	—	—			No anaemia	—		110 days..	17 days...	29 days.....	Marked anaemia.
41861	„	4	5 days	20 mg.	5+	3+	—	—	—	—	—	—	—		Slight anaemia. . .	—	—	110 days....	18 days . .	14 days....	Marked anaemia.
41875	41839 29/1/35	4	5 days	30 mg.	5+	+	—	—	—	—	—	—	—		No anaemia	—	—	110 days....	17 days.....	15 days....	Marked anaemia
41879	„	6	3 days	30 mg.	3+		—	—	—	—	—	—	—	—	No anaemia.........			110 days .			No reaction.
41839	Control	Untreated. Parasites present for 12 days. Severe anaemia developed.																			

work so that it was decided, in view of the observations quoted above, to determine the effect of the administration of neosalvarsan. For the work (Exp. S. 5414) sheep were selected whose previous history showed that they had not received any injections of fresh blood, in other words, it was hoped that only susceptible sheep would be selected. These animals were infected experimentally by the intravenous subinoculation of blood from a sheep in the acute stage of the disease. When parasites made their appearance in the blood stream in large numbers, doses of neosalvarsan varying from 5-45 mg. per kilo were injected intravenously. After administration of the drug, smears were examined at half hourly or hourly intervals for six hours and then daily examination was continued. As controls three sheep remained untreated.

The results are summarized in tabular form. (Table III.)

Results.—1. *Dose* 5 *mg. Neosalvarsan per Kilo Body Weight.*—From two out of three sheep parasites had disappeared after twenty-four hours: no indications of anaemia were observed, and no parasites reappeared for sixteen days, at which time smear examination was discontinued. In the case of the third sheep parasites persisted for sixteen days after treatment and slight anaemia developed.

2. *Dose* 7·5 *mg. per Kilo.*—Two sheep were treated. In both cases parasites persisted for twenty-four hours, but had disappeared after forty-eight hours and did not reappear for sixteen days, when examination was discontinued. Both animals developed severe anaemia.

3. *Dose* 15 *mg. per Kilo.*—Two sheep. In both cases the number of parasites had markedly decreased one hour after treatment, and had disappeared after two hours. Slight anaemia developed. The blood remained free from parasites for twenty and twenty-one days respectively when they reappeared. In spite of this relapse no anaemic changes developed.

4. *Dose* 30 *mg. per Kilo.*—Three sheep. In the case of two sheep, parasites were no longer demonstrable half an hour after treatment, and no anaemic changes developed; thirty-six and thirty-seven days later respectively parasites reappeared, persisted for eleven days and resulted in the production of slight anaemia. In the case of the third sheep the number of parasites in the blood gradually decreased up to the second hour after treatment by which time they had disappeared. No further observations could be carried out as the animal died from an intercurrent infection of heartwater three days later.

5. *Dose* 45 *mg. per Kilo.*—One sheep. Immediately after injection of the neosalvarsan parasites were present in large numbers, but they had disappeared within fifteen minutes. Slight anaemia developed. After an interval of twenty-nine days parasites reappeared and persisted for four days, but no evidence of anaemia was found in the smears.

6. *Controls Untreated.*—Three sheep. *Ep. ovis* could be demonstrated for eleven, twelve and fifteen days respectively and the anaemia produced was much more marked than in any of the treated sheep.

Conclusions.—From the results detailed above it may be concluded that in sheep a single intravenous injection of neosalvarsan in a dose of 5-7·5 mg. per kilo body weight has a well-defined parasiticidal action upon *Ep. ovis* resulting in disappearance of the parasites from the blood stream within forty-eight hours. As the dose is increased, the rapid specific action becomes more marked, the destructive action being roughly proportional to the dose, so that when 45 mg. per kilo is administered all parasites are eliminated from the blood stream within fifteen minutes. On the other hand, even a massive dose of 45 mg. per kilo is not a sterilising dose, since parasites reappeared approximately four weeks after treatment.

Discussion.—In practice the use of neosalvarsan is indicated as a specific for the treatment of Eperythrozoonosis, since even in small doses it has a marked beneficial effect upon the course of the disease, but it must be borne in mind that even a massive dose will not result in complete sterilization. This finding is in striking contrast with the results obtained in cases of *Bartonella canis* infection of dogs, where one-third of the maximum dose used in sheep, namely 15 mg. per kilo, was found to be a sterilizing dose. No opinion can be expressed as to the possible effect of repeated intravenous injections of the drug.

(ii) *Arseno-stibio Preparation Std.* 386 *B.*—In the treatment of *Bartonella muris* infection, Yoshiwara (1931) found the chemotherapeutic index of the antimony preparation stibosan to be 1:8, and Mayer, Borchardt and Kikuth (1927) demonstrated that of neosalvarsan to be 1:72. Uhlenhuth and Seiffert (1931) reported the remarkable properties of the antimony-arsenic compounds of Std. 283 and Std. 246, which have a chemotherapeutic index of 1:400. Dr. Hans Schmidt, who was responsible for the preparation of these two drugs, subsequently evolved a third, namely arseno-stibio compound 386 B which Kikuth (1932) and Uhlenhuth and Seiffert (1933) found to have the extremely high index of 1:3,500. These authors found that 18-24 days after treatment *Bartonella muris* reappeared in the blood stream in the majority of cases, but they were unable to determine whether this was the result of reinfection or was in the nature of a relapse, since, under the conditions of the experiments, natural transmission could not be excluded.

Bearing in mind the specific action of neosalvarsan on both *Bartonella muris* and *Ep. ovis* infections it was decided to investigate the action of the drug with the highest chemo-therapeutic index, namely arseno-stibio preparation Std. 368 B, in sheep. This experiment (S. 5572) was planned on lines similar to those of the analogous work on neosalvarsan, the results being summarized in tabular form in Table IV.

Results. 1. *Dose 5 mg. per Kilo Body Weight.*—Three sheep. From two sheep the parasites had disappeared two hours, from one sheep five hours after treatment, and in each case slight anaemia only developed. Two of the sheep showed a relapse thirty-five days later, the parasites persisting for seven and nine days respectively; no parasites were observed in the blood of the third sheep for a period of one hundred and ten days, when an immunity test was applied to all three by the subinoculation of infective blood. As a result of this immunity test the one sheep which did not show a relapse did not react, but one of the two sheep which did relapse proved immune, while in the other parasites appeared after seventeen days and persisted for seven days. After the immunity test daily smear examination was discontinued for forty-six days.

2. *Dose 10 mg. per Kilo.*—(Two sheep.) Parasites had disappeared from the blood of both animals in thirty to forty-five minutes respectively, and only a slight degree of anaemia developed. Over a period of one hundred and ten days no parasites were observed in either sheep, but then on immunity test reinfection became apparent on the seventeenth day, parasites persisted for ten and twenty-nine days respectively being accompanied by severe anaemia.

3. *Dose 20 mg. per Kilo.*—(Two sheep.) The results from the use of this concentration of the drug were practically identical with those obtained from the use of 10 mg. per kilo.

4. *Dose 30 mg. per Kilo.*—(Two sheep.) There was a marked decrease in the number of parasites fifteen minutes after treatment, and all had disappeared within thirty minutes in both cases. On immunity test after a lapse of one hundred and ten days, during which time the blood remained *Eperythrozoon* free, one sheep became reinfected after an incubation period of seventeen days the parasites persisting for fifteen days and producing severe anaemia, while the other was found to be immune.

Conclusions.—From the above results it is concluded that arseno-stibio preparation Std. 386 B. is indicated as a specific in the treatment of eperythrozoonosis in sheep. Used in the small dose of 5 mg. per kilo body weight, it may be relied upon to eliminate the parasites from the blood with great rapidity and to have a beneficial effect upon the subsequent course of the disease; only in a percentage of cases will complete sterilization be the outcome. The number of sheep used to test larger doses of the drug is too small to permit any generalization. It is significant, however, that 10 and 20 mg. per kilo resulted in sterilization in each of two groups of two sheep, so that failure of one animal, which received 30 mg. per kilo, to react on immunity test is inexplicable.

TABLE V.

Splenectomy of Ep. ovis *Carriers.*

D.O.B. No. of Sheep.	Date of splenectomy.	Total period of observation in days.	*Ep. ovis* appeared after.	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.	No. of relapses.	Interval in days of parasite free period.	No. of days during which *Ep. ovis* was present.	Nature of infection.	Remarks.
37429	8/2/35	448	6 days	12	6+	Severe anaemia	5	47 52 61 88 103	7 9 10 5 4	4+ 6+ 5+ 4+ 4+	Slight anaemia. Severe anaemia. Anaemia. Anaemia. Anaemia.
37862	8/2/35	150	6 days	11	5+	Severe anaemia	1	123	2	3+	Anaemia.

15. The Effect of Splenectomy.

After the removal of the spleen from two known carriers of *Ep. ovis* a reappearance of parasites in large numbers in the blood stream occurred on the sixth day. Severe anaemia and symptoms of a primary acute infection as seen in a susceptible animal then followed. Examination of daily blood smears from one sheep (37429) over a period of 448 days showed that during that period five separate relapses occurred, the last commencing fifteen months after splenectomy. This recrudescence of *Ep. ovis* after removal of the spleen from sheep harbouring a latent infection is further evidence of the significant rôle of the spleen in the mechanism of immunity to protozoan infections.

16. Immunity.

Many essential details on the immunity in this disease are lacking. This is chiefly due to the fact that the manner of natural transmission is not known and consequently in all experimental work the possibility of accidental infection cannot be eliminated with certainty. Nevertheless the available data from observations on splenectomized and non-splenectomized sheep together with the results obtained from the use of massive doses of specific chemotherapeutical drugs indicate that immunity must be regarded as a " labile infection " or " immunitas non sterilisans " which leads to an equilibrium between the parasite and the host.

No cases of auto-sterilization have been observed.

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Onderstepoort Journal of Veterinary Science and Animal Industry, Volume 9, Number 1, July, 1937.

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The occurrence of *Piroplasma pitheci* in a Vervet Monkey (*Cercopithecus aethiops cloetei* Roberts) in South Africa.

By W. O. NEITZ, Section of Protozoal and Virus Diseases, Onderstepoort.

THE occurrence of *P. pitheci* was recorded by Ross (1905) in several monkeys belonging to the genus Cercopithecus. The affected animals did not show any clinical symptoms apart from febrile reactions. Three years later the same preparations were re-examined by Nuttall and Graham-Smith, who stated that these parasites were of the *P. bigeminum* and *P. canis* type. Castellani and Chalmers (1908) described piroplasms in a monkey *Macacus pileatus* under the name *P. cellii.* A detailed account on the pathogenicity of *P. pitheci* in splenectomized and non-splenectomized monkeys belonging to the genus Cercopithecus and Macacus is given by Kikuth (1927). His experiments showed that non-splenectomized monkeys did not suffer from the infection, whereas in splenectomized animals numerous parasites appeared which produced severe anaemia, loss in condition and in one case death. Furthermore, he was able to show that trypan blue caused the parasites to disappear from the peripheral blood.

Schwetz (1933) described piroplasms resembling those described by Ross in two monkeys, one belonging to the genus Cercopithecus and the other to a species of Cercocoebus.

Through the kindness of Dr. A. D. Thomas of this Institute the writer obtained blood and spleen smears from an apparently healthy vervet-monkey which was shot on the farm Rossbach in the district Zoutpansberg in the Transvaal.

The films were stained with Giemsa. Piroplasms could be demonstrated in rare numbers identical with those described by Ross as *P. pitheci*. Only endoglobular forms were present. One, two and on one occasion four parasites per cell were seen. The majority were oval in shape, measuring from 1·5μ - 3μ in length by 1μ - 2μ in width. The ring forms measured from 2·5μ–3·0μ in diameter and the pear shaped forms 2·5μ long and 1·5μ wide. No actively dividing forms could be found. The nucleus of the elongated forms is situated at the broader end and in the ring forms consists of a band situated along a portion of the periphery. Slight anaemic changes were present in the nature of polychromasia and anisocytosis as well as a few normoblasts.

Table showing the different species of monkeys in which Piroplasms have been described.

Parasite.	Host.	Country.	Author.	Year.	Remarks.
P. pitheci	*Cercopithecus sp.*	Uganda....	Ross......	1905	Naturally infected cases.
P. cellii	*Macacus pileatus*	Ceylon....	Castellani and Chalmers	1908	The authors substituted the name *Theileria cellii.*
P. pitheci	*Cercopithecus sp.*	Africa......	Kikuth....	1927	Parasites appeared in the blood as result of splenectomy.
P. pitheci	*Macacus rhesus*	Germany..	Kikuth....	1927	This species of monkey found to be susceptible after receiving infected blood from a Cercopithecus monkey.
P. pitheci	*Cercopithecus sp.*	Belgium-Congo	Schwetz...	1933	Naturally infected case.
P. pitheci	*Cercocoebus sp.*	Belgium-Congo	Schwetz...	1933	Naturally infected case.
P. pitheci	*Cercopithecus aethiops Cloetei* Roberts	Low Country Transvaal, South Africa	Neitz......	1937	Naturally infected case.

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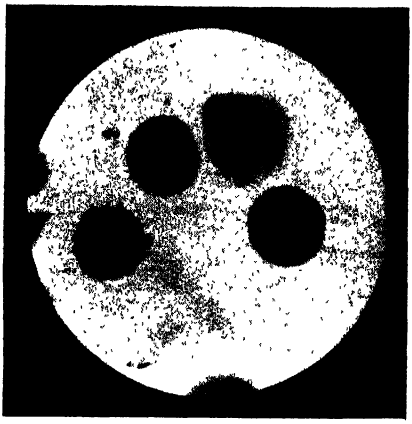

Fig. 1. *Piroplasma pitheci*, showing four parasites in an erythrocyte. ×1500.

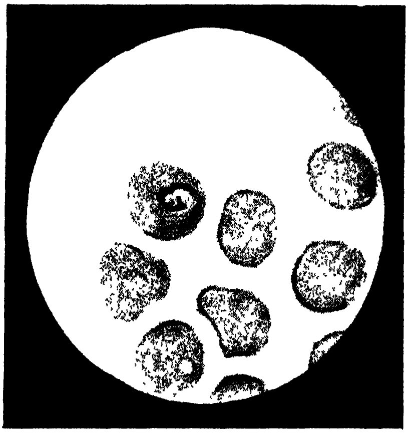

Fig. 2. *Piroplasma pitheci*, showing one large parasite in an erythrocyte. ×1500.

Section II.

Virus Diseases.

NEITZ, W. O. ... ... The transmission of heartwater to and from Blesbuck (*Damaliscus albifrons*) by means of the bont tick (*Amblyomma hebraeum*) 37

Onderstepoort Journal of Veterinary Science and Animal Industry, Volume 9, Number 1, July, 1937.

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Government Printer, Pretoria.

The Transmission of Heartwater to and from Blesbuck (*Damaliscus albifrons*) by means of the Bont-Tick (*Amblyomma hebraeum*).

By W. O. NEITZ, Section of Protozoology and Virus Diseases, Onderstepoort.

INTRODUCTION.

IN 1933 and 1935 the writer reported on the transmission of heartwater to two species of South African antelopes namely the blesbuck (*Damaliscus albifrons*) and the black wildebeest (*Conochaetes gnu*). No visible clinical symptoms could be detected in these animals, but the presence of the " virus " could be demonstrated by subinoculating blood into susceptible sheep. These results prompted further studies on the transmission of this disease to antelopes by the bont-tick (*Amblyomma hebraeum*). The outcome of this work is published because it is believed that the experiments illustrate that heartwater can exist under natural conditions in the absence of domestic ruminants. Furthermore, this knowledge is of extreme practical importance and must serve as a basis for the promulgation of any prophylactic measures which aim at the eradication of heartwater.

Through the kindness of the Provincial Administration of the Orange Free State this Institute obtained several blesbuck from the Summerville Game Reserve, an area where heartwater is not known to exist. The animals were kept in a camp comparatively free of ticks. When the experiments were started the blesbuck were wild and difficult to handle. The temperature of the first animal could therefore not be taken. Later on the animals became more docile and it was possible to take the temperature twice a day from the second blesbuck.

THE BREEDING AND FEEDING OF TICKS.

In the literature numerous references to this subject are given. The methods in these experiments have been modified to some extent with the object of reducing the period of hatching of the eggs and moulting of the various stages. For this purpose the *A. hebraeum* ticks were kept in a room of constant temperature of 26° C. and a relative humidity of 80 per cent. Under these conditions the ticks thrive well and their life cycle can be completed within six months.

Another improvement that was necessary is the method of handling and feeding, since during such operations one has to consider the danger of ticks escaping. To overcome this difficulty bags having the shape of an ear are made from thin but strong linen. With the aid of a funnel and by careful manipulation the ticks are shaken into the bottom of the bag, the tip of which is tied off with tape in order to confine them there. The bag is then drawn over the ear and the open end fixed to the base of the ear by means of adhesive paste. When the paste has dried the tape is removed and the ticks are thereby liberated into a confined space. As a further precaution larger stout calico bags fitted with several lengths of tape are placed on both ears, to prevent damage to the inner bag. (See Figs. 1, 2 and 3.)

The bags are left undisturbed for three days. When ticks have to be collected the outer bag is removed. An incision about 8 to 10 cms. is made in the inner bag and the ticks can be examined. After collection the incision is sewn up and the outer bag placed back into position.

This system has been found to be satisfactory and if carefully carried out one can account for practically every tick that has been placed on the animal.

TICK TRANSMISSION.

For the sake of convenience the details of the experiments are appended in Tables I-III.

Experiment No. 1 (*S.* 5527).

Object.—To infect *Amblyomma hebraeum* larvae with heartwater.

Method.—(*a*) Virulent heartwater blood was injected intravenously into a susceptible sheep 45733. (*b*) On the fourth day after injection the ears of the sheep were infested with three hundred larvae batch 1416A.

Result.—The ticks fed readily and 225 engorged larvae were collected from the sheep during the reaction. The rest of the ticks had not attached and were found dead in the bag.

Conclusion.—No satisfactory explanation can be given why a number of apparently healthy ticks die when they are placed on the ears of an animal. In some tick feeding experiments carried out at Onderstepoort up to 50 per cent. of larvae have been found dead twenty-four hours after infestation.

The engorged larvae were allowed to moult and were used in the subsequent experiments.

Experiment No. 2 (*S.* 6020).

Object.—To ascertain whether the nymphae that fed as larvae on sheep 45733 in experiment No. 1 are able to transmit heartwater to a sheep.

FIG. I.—Blesbuck 47081 illustrating the method of fixing inner tick bags to an ear. For protection larger loosely fitting calico bags are placed over the inner bag.

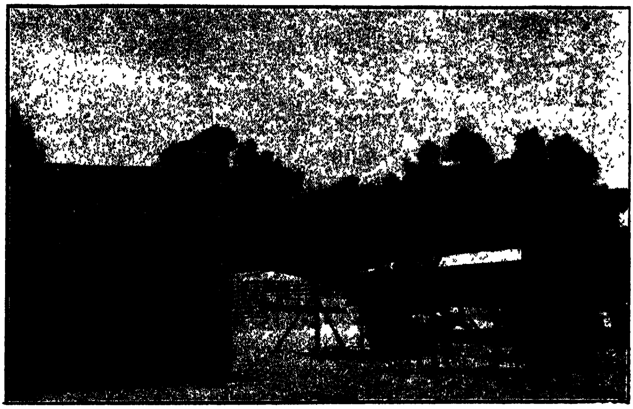

FIG. II.—Blesbuck 47081 roaming freely in the camp with outer bag for protection attached.

FIG. III.—Sheep with inner tick bag attached.

Method.—The ears of a susceptible sheep 45872 were infested with approximately 18 nymphae batch 1416Ab.

Result.—The sheep reacted to heartwater and died. All the ticks had dropped by the 8th day.

Conclusion.—The ticks picked up the infection as larvae and were able to transmit heartwater as nymphae. This batch of ticks is considered suitable for further transmission work.

Experiment No. 3.

Object.—To transmit heartwater to a blesbuck by infesting the animal with known infected ticks.

Method.—Approximately 50 infected *A. hebraeum* nymphae batch 1416Aa that fed as larvae on sheep 45733 in experiment No. 1 were allowed to feed on the left ear of blesbuck No. 47081.

Result.—The ticks attached readily, but the feeding period was considerably longer than in the case of sheep 45872 in experiment No. 2. Ten ticks had not attached and were found dead in the bag. Forty engorged specimens were collected between the 8th to the 20th day, whereas in sheep 45872 the ticks dropped from the 6th to the 8th day.

Conclusion.—The feeding in the case of the sheep was carried out in a stable whereas in the case of the blesbuck in a camp. No satisfactory explanation can be given why the ticks took longer to engorge on the latter animal. An important factor may be the temperature which is more constant in a stable than in a camp.

Experiment No. 4.

Object.—(*a*) To ascertain whether nymphae have transmitted heartwater to blesbuck 47081. (*b*) To ascertain whether ticks will infect themselves while feeding on this blesbuck.

Method.—(*a*) Blood from blesbuck 47081 was injected at various intervals after tick infestation into susceptible sheep. (*b*) 14 days after infesting the left ear 100 larvae batch 1416D. and 20 clean nymphae batch 1396Aa were placed on the right ear of the blesbuck.

Result.—(*a*) It was possible to demonstrate heartwater " virus " by subinoculating blood into sheep from the 28th to the 62nd day after tick infestation, i.e. for a period of 35 days. Sheep injected before this period did not react to heartwater.

The blesbuck died 70 days after tick infestation. Blood was collected after death but did not produce heartwater when injected into two susceptible sheep. The post-mortem examination of the animal showed hydrothorax, hydropericard oedema of the lungs and a few *Haemonchus contortus* in the abomasum. *Rickettsia ruminantium* could not be demonstrated in the smears prepared from the intima of the jugular vein.

(*b*) (i) Eighty-seven engorged larvae detached from the 13th to the 20th day The rest had died. (ii) Fifteen engorged nymphae were collected from the 14th to the 17th day, the others had not attached and died. These ticks were allowed to moult and fed in their next stage on sheep in order to establish their infectivity.

Conclusion.—The incubation period of heartwater in blesbuck 47081 was considerably longer (approximately 28 days) than that of sheep 45872 (12 days). This probably stands in relation to the longer period taken by the ticks to engorge on the former animal.

During the time that the clean ticks were feeding on the blesbuck the presence of heartwater virus could be demonstrated by blood subinoculation. The larvae and nymphae therefore had every opportunity of infecting themselves.

Experiment No. 5.

Object.—To ascertain whether the larvae and nymphae infected themselves while feeding on blesbuck 47081.

Method.—(*a*) The nymphae batch 1416Da were allowed to feed on sheep 45980. (*b*) The adults batch 1396Aa1 were allowed to feed on sheep 46069.

Result.—(*a*) Sheep 45980 reacted to heartwater and recovered. (*b*) Sheep 46069 reacted to heartwater and died.

Conclusion.—The larvae and nymphae picked up the infection while feeding on blesbuck 47081 and transmitted heartwater to sheep when fed as nymphae and adults respectively.

Experiment No. 6.

Object.—To ascertain whether adult ticks that transmitted heartwater as nymphae to blesbuck 47081 retain their infection.

Method.—Adult *A. hebraeum* ticks batch 1416Aa1 were allowed to feed on sheep 45874.

Result.—The male ticks attached readily but the females only three days later. They detached from the 11th to the 17th day. The sheep reacted to heartwater and was killed *in extremis*.

Conclusion.—The adult ticks retained their infection.

Experiment No. 7 (*S.* 6104).

This experiment was carried out with the object of obtaining further information on the transmission of heartwater to a blesbuck by means of ticks.

Object.—To transmit heartwater to a blesbuck by infesting the animal with known infected ticks.

TABLE I.

The Feeding of Amblyomma hebraeum *on the Blesbuck.*

D.O.B. No. of animal and date of infestation.	Infested with batch.	Stage.	History of ticks.	Object.	Ticks drop: (a) Number of days after infestation. (b) Number of ticks collected.	Result.
Bb. 47081 25/8/36	1416 Aa	Nymphae	Ticks fed as larvae on a heartwater reacting sheep 45733	To transmit heartwater to the blesbuck	(*a*) 8, 10, 13, 14, 16, 18, 20 (*b*) 4, 1, 20, 10, 2, 1, 1	Animal showed a gradual loss of condition for a period of 7 weeks and died 3/11/36. No Rickettsia could be found in the intima smears from the jugular vein.
Sh. 45872 25/8/36	1416 Ab	Nymphae	Ticks fed as larvae on a heartwater reacting sheep 45733	To show that the batch of ticks are infected with heartwater	(*a*) 6, 7, 8 (*b*) 7, 7, 2	Reacted to heartwater 12 days after infestation and died 15/9/36
Bb. 47081 8/9/36	1416 D	Larvae	The progeny of adults collected at Pretoria North on 7/1/36	To infect larvae with heartwater	(*a*) 13, 14, 15, 16, 17, 20 (*b*) 2, 30, 10, 10, 25, 10	During the time that the larvae were feeding the presence of heartwater "virus" could be demonstrated by subinoculation into susceptible sheep.
Bb. 47081 8/9/36	1396 Aa	Nymphae	Larvae fed on a guinea pig	To infect nymphae with heartwater	(*a*) 14, 15, 16, 17 (*b*) 2, 5, 2, 6	During the time that the nymphae were feeding, the presence of heartwater "virus" could be demonstrated by subinoculation into susceptible sheep.
Sh. 45980 6/11/36	1416 Da	Nymphae	Fed as larvae on blesbuck 47081	To ascertain whether these ticks became infected with heartwater	(*a*) 5, 8, 11 (*b*) 1, 10, 2	Reacted to heartwater 12 days after tick infestation and recovered. On testing the immunity the sheep was found to be immune.
Sh. 46069 13/11/36	1396 Aa1	Adults	Fed as nymphae on blesbuck 47081	To ascertain whether these ticks became infected with heartwater	(*a*) 10, 13 (*b*) 1, 2	Reacted to heartwater 17 days after tick infestation and died 6/12/36.
Sh. 45874 13/11/36	1416 Aa1	Adults	Transmitted heartwater to the blesbuck 47081 in the nymphal stage	To ascertain whether these ticks will retain the infection and pass it off in the adult stage	(*a*) 11, 12, 13, 17 (*b*) 2, 2, 2, 1	Reacted to heartwater 15 days after tick infestation. Animal destroyed in extremis.
Bb. 47083 21/12/36	1416 Ac	Nymphae	Ticks fed as larvae on a heartwater reacting sheep 45733	To transmit heartwater to the blesbuck	(*a*) 7, 8, 10 (*b*) 5, 6, 3	Animal showed a gradual loss of condition for a period of 13 weeks and died 22/3/37. No Rickettsia could be found in the intima smears from the jugular vein. From the 17 to 26 day after tick infestation, heartwater "virus" could be demonstrated by subinoculation into sheep.

Bb. = Blesbuck. Sh. = Sheep.

TABLE II.

The Demonstration of Heartwater "Virus" in Blesbuck 47081 *by Subinoculation in Sheep.*

D.O.B. No. of animal.	Dose of blood i.v.	Number of days after tick infestation.	Date.	Incubation period in days.	Result.
43761	25 c.c.	14	8/ 9/36	—	No heartwater reaction.
42735	10 c.c.	20	14/ 9/36	—	No heartwater reaction.
45813	10 c.c.	20	14/ 9/36	—	No heartwater reaction.
42837	10 c.c.	24	18/ 9/36	—	No heartwater reaction.
43659	10 c.c.	24	18/ 9/36	—	No heartwater reaction.
46020	10 c.c.	28	22/ 9/36	11	Reacted to heartwater and died 16/10/36.
46123	10 c.c.	28	22/ 9/36	13	Reacted to heartwater and died 13/10/36.
46041	10 c.c.	31	25/ 9/36	10	Reacted to heartwater and recovered. On testing the immunity found to be immune.
46092	10 c.c.	31	25/ 9/36	10	Reacted to heartwater and died 19/10/36.
46228	10 c.c.	42	6/10/36	9	Reacted to heartwater and died 28/10/36.
46434	10 c.c.	42	6/10/36	10	Reacted to heartwater and died 21/10/36.
45974	10 c.c.	52	16/10/36	13	Reacted to heartwater and recovered. On testing the immunity found to be immune.
46340	10 c.c.	52	16/10/36	8	Reacted to heartwater and died 4/11/36.
43433	10 c.c.	62	26/10/36	12	Reacted to heartwater and recovered. On testing the immunity found to be immune.
46085	10 c.c.	62	26/10/36	10	Reacted to heartwater and recovered. On testing the immunity found to be immune.
46353	10 c.c.	70	3/11/36	—	No heartwater reaction. On testing the immunity found to be susceptible.
46728	10 c.c.	70	3/11/36	—	No heartwater reaction. On testing the immunity found to be susceptible.

Method.—(*a*) Approximately 20 infected *A. hebraeum* nymphae batch 1416Ac collected from sheep in experiment No. 1 were allowed to feed on the ear of blesbuck 47083. This animal was docile and the temperature was taken twice daily. (*b*) Blood was injected at various intervals after tick infestation into susceptible sheep.

Result.—(*a*) The ticks fed readily and 14 specimens detached from the 7th to the 10th day.

The temperature did not show any abnormal variation. There was a gradual loss of condition in this animal which died eight weeks after the last day on which heartwater " virus " was demonstrated. At autopsy the blesbuck was found to be suffering from bronchopneumonia. In addition there was a light Haemonchus and Dictyocaulus infection.

(*b*) The presence of heartwater " virus " could be demonstrated from the 17th to the 26th day but not on the 31st day after tick infestation. Only one out of two sheep reacted in each group.

Conclusion.—It was possible to transmit heartwater to blesbuck 47081 by means of infected *A. hebraeum* nymphae. Attention is drawn to the fact that only three out of six sheep reacted when injected with blood during the time while the blesbuck harboured heartwater " virus ". From this observation it is not clear what factors control the infectivity of blood.

The incubation period was approximately 17 days whereas in the previous blesbuck, 47083, it was approximately 28 days.

TABLE III.

The Demonstration of Heartwater " Virus " in Blesbuck 47083 *by Subinoculation into Sheep.*

D.O.B. No. of animal.	Dose of blood i.v.	Number of days after tick infestation.	Date.	Incubation period in days.	Result.
43556	10 c.c.	17	7/1/37	—	No heartwater reaction. On testing the immunity found to be susceptible.
43755	10 c.c.	17	7/1/37	11	Reacted to heartwater and recovered. On testing the immunity found to be immune.
45736	10 c.c.	23	13/1/37	12	Reacted to heartwater and died 1/2/37.
45747	10 c.c.	23	13/1/37	—	No heartwater reaction. On testing the immunity found to be susceptible.
45725	10 c.c.	26	16/1/37	11	Reacted to heartwater and died 3/2/37. *Rickettsia ruminantium* could be demonstrated in the smears prepared from the intima of the jugular smears.
45891	10 c.c.	26	16/1/37	—	No heartwater reaction. On testing the immunity found to be susceptible.
42832	10 c.c.	31	21/1/37	—	No heartwater reaction. On testing the immunity found to be susceptible.
44525	10 c.c.	31	21/1/37	—	No heartwater reaction. On testing the immunity found to be susceptible.

DISCUSSION.

Amblyomma hebraeum is widely distributed in the Union of South Africa and is found chiefly in the Lowveld, Northern and Eastern Transvaal, Natal and Eastern Cape Province. It has been recorded from Bechuanaland. Not only does this species of tick parasitize domestic stock, but Bedford (1932 and 1936) records its presence from several different species of antelopes mentioned in Table IV and also from other wild animals.

TABLE IV.

Species of Antelopes from which A. hebraeum *has been recorded.*

Zoological name.	Vernacular name.	Stage.	Locality.
Gorgon taurinus............	Blue Wildebeest	Adults	Umfolosi Game Reserve, Natal.
Nyala angasi..............	Nyala	Adults	Ubombo Flats, Zululand.
Syncerus caffer............	African Buffalo	Adults	Umfolosi Game Reserve, Natal.
Sylviacapra grimmi.........	Duiker	Adults & Nymphae	Umfolosi Game Reserve, Natal.
Strepsiceros strepsiceros......	Koodoo	?	South Africa.
Tragelaphus sylvaticus......	Bushbuck	?	South Africa.
Aepyceros melampus........	Impala	Adults	Swaziland.

Since ticks that become infected with heartwater as larvae retain their infection until the adult stage it is believed that antelopes and possibly other wild animals are capable of spreading infected ticks over considerably large areas.

The experiments mentioned in this paper show conclusively that infected ticks can transmit heartwater to a blesbuck and that clean ticks in their turn are capable of infecting themselves when they are allowed to feed on such an infected animal. Under natural conditions therefore the same may happen.

From observations it is known that the bont tick(*A. hebraeum*) may take a very long time to complete its life cycle. When conditions are ideal the period from the larvae to the adult stage can be as short as six months and under less favourable conditions the period is extended to over two years. During this time several susceptible animals can be born. It is therefore possible that in areas where there are no domestic animals and where infected ticks have to rely chiefly on antelopes and their susceptible progeny as hosts heartwater can be maintained.

Up to the present heartwater has not been demonstrated in antelopes living under natural conditions. During the investigations on trypanosomiasis in antelopes a large number of blood subinoculations was made from these animals into domestic ruminants. It would be interesting to know whether any of the workers observed cases of heartwater in the injected animals while carrying out their studies. Naturally if immune animals were used in their experiments heartwater would not have been found.

SUMMARY.

(1) The transmission of heartwater to two blesbuck is discussed.

(2) Infected *A. hebraeum* nymphae are capable of transmitting heartwater to the blesbuck.

(3) Heartwater could be demonstrated by blood subinoculation into susceptible sheep for a period of 35 days in one blesbuck and for 9 days in another.

(4) The virulence of heartwater virus did not change by passage.

(5) Infective nymphae do not loose their infection but retain it to the adult stage.

(6) Clean larvae will pick up infection for transmission as nymphae.

(7) Clean nymphae will pick up infection for transmission as adults.

(8) The technique of tick breeding and feeding is briefly discussed.

(9) Both blesbuck died, one 13 and the other 7 weeks after tick infestation.

(10) The significance of these experiments is discussed.

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Section III.

Bacteriology.

STERNE, MAX ... ... The effects of different carbon dioxide concentrations on the growth of virulent anthrax strains. Pathogenicity and immunity tests on guinea-pigs and sheep with anthrax variants derived from virulent strains 49

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The Effects of Different Carbon Dioxide Concentrations on the Growth of Virulent Anthrax Strains.

Pathogenicity and Immunity Tests on Guinea-pigs and Sheep with Anthrax Variants derived from Virulent Strains.

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INTRODUCTION.

IN a previous paper (Sterne, 1937) fully virulent anthrax stains were shown to grow smooth mucoid if cultured on 50 per cent. serum agar in an atmosphere of about 65 per cent. carbon dioxide. Such cultures immediately gave rise to rough non-capsule-producing avirulent dissociants. A similar phenomenon has been described by Schaeffer (1936) who found that anthrax strains grew mucoid on coagulated serum and produced rough variants. He cultivated these on serum for several generations and eventually found that they became reduced in virulence or avirulent. The ability to produce capsules was also lost. Sometimes these rough variants still killed and produced capsules. Therefore Schaeffer postulated a stable and an unstable variety of rough. This assumption seems unnecessary in view of the difficulty of ensuring the absolute homogeneity of the selected variants. This difficulty is inherent in all variation work unless single cell isolation technique is used, and Schaeffer's use of coagulated serum rendered his work liable to this source of error. Inspissated serum was tried previously (Sterne 1937) without much success. Fully virulent strains did not grow very mucoid, and moreover were so markedly proteolytic that the surface of the medium became semi-liquid. This rendered the isolation of " pure " variants exceedingly difficult. Stamatin (1934) grew virulent anthrax strains in defibrinated horse blood until mucoid colonies appeared. These produced rough, uncapsuled and avirulent dissociants. Stamatin and Stamatin (1936) produced immunity in rabbits with one of their avirulent strains. Both Stamatin's and Schaeffer's work involved considerable manipulation of their cultures.

Objects.

The results previously obtained with variants developed on serum agar in carbon dioxide were encouraging enough to warrant more extensive and critical experiments. These were carried out to discover the effects of varying the carbon dioxide concentration on virulent strains grown on serum agar and to examine the influence of the different concentrations on the development of mucoid growth, the production of avirulent roughs and the immunizing power of these roughs.

Technique.

The medium used was 50 per cent. horse serum nutrient agar. This was put up in Mason's tubes (Mason 1933) and partially dried in the incubator for 24 hours. Thereafter, the fluid liberated by syneresis was removed and the tubes were then ready for use. The inoculation was always done on about one square centimetre of surface and incubation carried out in anaerobic jars of 1,900 ml. capacity to which the required amount of carbon dioxide was added. No satisfactory method of keeping the carbon dioxide pressure constant was devised and therefore the results must be interpreted for the particular system used here: that is, each jar of 1,900 ml. contained one Mason's tube inoculated on one square centimetre of surface. Whenever incubation lasted more than 24 hours the jars were opened daily and the carbon dioxide tension adjusted. The method of adding carbon dioxide was described in a previous paper.

Mason's tubes were inoculated with the different virulent strains and incubated in various carbon dioxide concentrations. The results are tabulated below.

The following symbols are used to describe the character of the growth:—

SM denotes a colony with a rough edge and slightly mucoid surface.

*S*M denotes a colony as above, but with a more mucoid surface.

SM denotes a colony with a smooth edge and completely mucoid surface.

S*M* denotes a colony with a very mucoid surface, smooth edges and a tendency to flow.

S**M** denotes a colony that can be drawn into long threads when touched with a needle. The growth may be as much as half a centimetre thick.

The extent of development of rough dissociants is shown thus:—

+ denotes minute rough projections too small to be picked.

++ denotes well developed rough wedges or outgrowths, easy to pick.

+++ denotes extensive well defined rough projections from the smooth edge of the colony.

++++ denotes a broad ring of rough growth entirely surrounding a mucoid centre.

Occasionally, in the tables, the symbols SM or S*M* appear associated with the symbol ++++. This is really in conflict with the scheme, but it signifies a very well developed mucoid centre entirely surrounded by a sharply demarcated rough zone. In these cases it was obvious that the ring of rough growth was due to a running together of rough outgrowths. The tables show that such a combination of symbols is only shown after 48 hours, whereas the colonies at 24 hours showed smooth edges between the rough dissociants.

R denotes completely rough growth.

S denotes completely smooth growth.

RS denotes intermediate or rough-smooth growth and the relative preponderance of one or the other factor is indicated thus *R*S or R*S*.

The virulence tests in Tables I to V were done on guinea-pigs. Three pigs were used for each variant and each received subcutaneously one-third of an agar slope of the particular variant indicated in the table.

EXPERIMENTS.

1.—*The effect of different carbon dioxide concentrations on virulent strain XXVIII.*

This strain was isolated sixteen years ago and is still very virulent for sheep.

TABLE I.

Tube No.	% CO_2.	Type of growth after.				Virulence Test.							
		24 hrs.	48 hrs.	72 hrs.	96 hrs.	Subbed after hrs.	Type of variant subbed.	Death : hours after inoculation.					
								20	40	60	80	100	
A	0	RS	SM	—	—		—						
B	0	R*S*	*S*M	—	—	48	rough edge		† † †				
C	0	RS	RS	—	—								
D	5	*S*M +	SM ++	—	—								
F	10	*S*M	S*M*	—	—								
G	10	*S*M	S*M*	—	—	48	S*M*		† † †				
		+	+++	—	—	48	R (+++)			†	†		/

TABLE I—(*continued*).

Tube No.	% CO_2.	Type of growth after.				Virulence Test.		Death : hours after inoculation.					
		24 hrs.	48 hrs.	72 hrs.	96 hrs.	Subbed after hrs.	Type of variant subbed.	20	40	60	80	100	
H	15	S*M*	S*M*	—	—	48	S*M*		† †	 †			
		+	++	—	—	48	R (++)						/ / /
I	20	S*M*	S*M*	—	—	48	S*M*		† † †				
			+	—	—	48	R (+)		† †	 †			
J.	30	S*M*	SM	—	—	48	SM	†	† †				
			++	—	—	48	R (++)						/ / /
K	40	S*M*	SM	—	—	48	SM		† † †				
			++	—	—	48	R (++)						/ / /
L	50	SM ++	SM +++	—	—								
M	50	SM	S*M* ++	—	—								
N	65	SM	SM	SM	SM	72	SM		† † †				
			+	++	+++	72	R (++)						/ / /
						96	SM		† † †				
						96	R (+++)						/ / /
O	65	SM	SM +	—	—								
P	75	SM	SM +	—	—								

† = Guinea pig, died of anthrax. / = Survived.

Thus mucoid growth was best in carbon dioxide concentrations between 10 and 50 per cent. and dissocation was most active over approximately the same range. Subcultures from smooth mucoid growth in 10 to 65 per cent. carbon dioxide were virulent whereas roughs obtained in the same concentrations were reduced in virulence or avirulent. A subculture from the rough edge of B in 0 per cent. CO_2 was fully virulent. This confirmed previous observations.

2.—*To test the effects of different carbon dioxide concentrations on virulent strain XXXIII.*

This strain was isolated from a goat which died of anthrax, naturally acquired, six weeks previously.

TABLE II.

Tube No.	% CO_2.	Type of growth after.		Virulence Test.							
				Subbed after. Hrs.	Type of variant subbed.	Death : Hours after inoculation.					
		24 hrs.	48 hrs.			20	40	60	80	100	
A	5	RS	--								
B	10	SM	SM	24	SM		† ††				
		++	++++	24	R (++)						/ / /
				48	R (++++)						/ / /
C	20	SM ++	R ++++								
D	30	SM	R	24	SM		† † †				
		++	++++	24	R (++)		†	††			
E	30	SM ++	RS ++++								
F	40	SM ++	—								
G	50	SM +	SM ++++								
H	50	SM ++	SM ++++								
I	60	RS +++	RS ++++								
J	65	SM +	SM ++++								
K	70	SM ++	RS ++++								

Mucoid growth was best maintained in carbon dioxide concentrations between 20 and 50 per cent., but even at best was not marked. By the second day the mucoid character had almost disappeared. The guinea-pig tests showed that this was due to the rapid development of rough avirulent variants swamping the mucoid growth.

3.—*The effect of different concentrations of carbon dioxide on strain XXIV.*

This strain was isolated four months previously from a bovine which had died of anthrax.

TABLE III.

Tube No.	% CO_2.	Type of growth after.		Virulence Test.							
		24 hrs.	48 hrs.	Subbed after. Hrs.	Type of variant subbed.	Death : Hours after inoculation. 20	40	60	80	100	
A	0	RS	RS								
B	5	SM +++	—								
C	10	SM +++	SM ++++								
D	20	SM	RS	24	SM		† † †				
		+++	++++	24	R (+++)	All guinea pigs died in a week, showed marked oedema but no capsuled bacilli. Test repeated with first sub-culture (below).					
				1st sub of R	culture (+++)						/ / /
E	20	SM +++	—								
F	20	SM +++	RS ++++								
G	30	SM +++	—								
H	30	SM	RS	24	SM		† † †				
		+++	++++	24	R (+++)		† †				/
I	30	SM +++	—								
J	30	SM	RS								
		+++	++++	48	R (++++)						/ / /

TABLE III—(*continued*).

Tube No.	% CO_5.	Type of growth after.		Virulence Test.						
		24 hrs.	48 hrs.	Subbed after. Hrs.	Type of variant subbed.	Death : Hours after inoculation.				
						20	40	60	80	100
K	40	SM	RS							
		+++	++++	48	R (++++)					/ / /
L	50	RS	RS							
		+++	++++	48	R (++++)					/ / /
M	60	R*S*	RS							
N	60	SM	—							
O	80	R*S* ++	R*S* ++							

Strain XXIV never produced completely mucoid growth. The centre of the culture showed a slightly mucoid surface in carbon dioxide concentrations from 5 to 40 per cent., but usually this was gone at 48 hours. No other virulent strain had behaved in this way and therefore attempts were made to obtain more profuse mucoid growth by using different proportions of serum or whole blood. All were unsuccessful. However, the protocols of the guinea-pig tests show that the rough growth was not due to an inability of the virulent strain to become mucoid, but rather to the rapid proliferation of avirulent rough variants; because subcultures from the edges of the rough colonies were usually avirulent.

4.—*To test the effect of different carbon dioxide concentrations on virulent strain* XXXIV.

This strain was isolated from the hide of a bovine seven days previously.

TABLE IV.

Tube No.	% CO_2.	Type of growth after.		Virulence Test.							
		24 hrs.	48 hrs.	Subbed after. Hrs.	Type of variant subbed.	Death : Hours after inoculation.					
						20	40	60	80	100	200
A	0	—	*R*S								
B	0	*R*S	*R*S								
C	10	SM	*S*M	24	SM		† † †				
		+++	++++	24	R (+++)						/ / /

TABLE IV—(*continued*).

Tube No.	% CO_2.	Type of growth after.		Virulence Test.								
		24 hrs.	48 hrs.	Subbed after. Hrs.	Type of variant subbed.	Death : Hours after inoculation. 20	40	60	80	100	200	
D	10	*S*M ++	SM ++++									
E	20	*S*M +	SM +++									
F	30	*S*M	S*M*	24	*S*M		† † †					
		++	+++	24	R (++)							/ / /
				48	S*M*		† † †					
				48	R (+++)						†	/ /
G	30	*S*M	S*M*	48	S*M*		† † †					
			+++	48	R (+++)							/ / /
H	40	*S*M ++	SM ++++									
I	50	SM +	SM ++++									
J	60	RS	*S*M									
K	60	*S*M +	*S*M ++									
L	70	S*M*	S*M*									
		+	++	48	R (++)							† † /
M	75	SM	*S*M	48	*S*M		† † †					
			++	48	R (++)							/ / /
N	80	*S*M	SM +									

This strain was more mucoid in carbon dioxide concentrations between 20 and 70 per cent. and rough dissociants were more freely produced in 10 to 50 per cent. concentrations. All the smooth mucoid variants tested were virulent for guinea-pigs and the roughs avirulent or reduced in virulence.

5.—*To test the effects of different carbon dioxide concentrations on virulent strain XXXV.*

This strain was isolated two days previously from a bovine dead from naturally acquired anthrax.

TABLE V.

Tube No.	% CO_2.	Type of growth after.		Virulence Test.						
		24 hrs.	48 hrs.	Subbed after. Hrs.	Type of variant subbed.	Death : Hours after inoculation.				
						20	40	60	80	100
A	10	SM	SM							
		+	++++	48	R (++++)					/ / /
B	20	SM	SM							
			++	48	R (++)					/ / /
C	30	SM	SM	48	SM		† † †			
			++	48	R (++)					/ / /
D	60	RS	RS †							
E	80	RS	RS	48	rough edge		† † †			

Thus mucoid growth was good in carbon dioxide concentrations between 10 and 30 per cent. In 60 and 80 per cent. there was no mucoid development nor production of rough dissociants. Avirulent roughs were obtained in all the cultures grown in concentrations up to 30 per cent., whereas the non-mucoid culture in 80 per cent. CO_2 had lost no virulence. Culture B was rather interesting. The avirulent variant grew as a thin rough film (phantom colony, Nungester 1929) and within this film rough non-phantom colonies developed. This process recurred with every subculture of the phantom. Neither variant killed.

DISCUSSION.

Five virulent strains were grown in different carbon dioxide concentrations and examined for their ability to grow mucoid and to produce rough avirulent dissociants. Mucoid growth was found to depend partly on the strain and partly on the carbon dioxide concentration. Sometimes the mucoid growth was not persistent due to the rapid proliferation of avirulent rough variants. Strains tested in 0 per cent. and 80 per cent. carbon dioxide did not grow mucoid and produced no avirulent dissociants. The optimum concentrations of carbon dioxide for obtaining avirulent roughs quickly appeared to lie between 10 per cent. and 30 per cent.

In general, mucoid growth was not as profuse as in the experiments described in the previous paper. This was probably due to the use of a different batch of medium.

In this paper and in a previous paper it was shown that all virulent anthrax strains dissociate in an apparently orderly manner from the time they are isolated. In the particular cases investigated there was a continuous variation in the direction of uncapsuled and avirulent types. The regularity of the process makes it difficult not to entertain the hypothesis that this may be a constant feature of growth in anthrax even under natural conditions and that it may have a bearing on epidemiological and immunological problems in nature.

An interesting aspect of the investigation was the variation in the dissociation rates of the different virulent strains. This carries the possibility that there are fundamental differences between virulent strains and that these differences may play a determining role in, for example, the persistence of an infection in the field.

COMPARATIVE IMMUNITY TESTS ON GUINEA-PIGS AND SHEEP WITH AVIRULENT VARIANTS OBTAINED IN DIFFERENT CONCENTRATIONS OF CARBON DIOXIDE.

It was shown previously that sheep could be immunized with avirulent rough variants. The present series of experiments was designed to examine the possibility of using avirulent rough variants as vaccines. These dissociants have obvious advantages over the ordinary attenuated strains: they are avirulent; they produce 99 per cent, free-lying spores in 3 to 4 days; and they can be obtained from virulent strains in 24 to 48 hours.

Parallel experiments were done on guinea-pigs and sheep to see if there was a relation between the degree of immunity produced in these species. Sheep have always been used at Onderstepoort as the final laboratory test animal, so that considerable saving would result if preliminary guinea-pig tests could eliminate strains likely to be unsuccessful as vaccines, before the more expensive sheep test was used.

The majority of the strains now tested on sheep produced poor immunity in guinea-pigs. These strains were tested purposely, because it was as important to know the worst results to be expected, as to know the best.

The strains used for immunization were isolated in the experiments noted in Tables I-V, with the exception of strain XXII A_2 which was isolated some months before. The guinea-pigs tested (except with strain XXII A_2) were those which survived in the experiments noted in Tables I to V. Sheep were immunized with 1/300 to 1/500 dilution of a fully sporulated agar slant (1·5 × 7 cm.) of the avirulent strains. This dose was somewhat smaller than that used in the ordinary pasteur II type of vaccine prepared here for issue. For the sake of comparison, tests on the routine Pasteur II type vaccine were done at the same time as the tests on the avirulent strains, and these results are also shown in the tables. The 0·01 dilution of the Pasteur II is slightly more than 1:300 agar slant.

The virulent test dose for guinea-pigs was ¼ to ⅛ of an agar slant (1·5 × 7 cm.) of strain II Ad. This was the smooth mucoid strain used in previous tests (Sterne 1937), but its virulence had since been considerably enhanced by serial passage through guinea-pigs.

It now always killed unprotected guinea-pigs in 15 to 40 hours, but the majority were dead by the 24th hour. In the first experiment (Strain XXII A_2) the test strain II Ad had not yet been exalted.

The test dose for sheep was a sporulated glycerine-saline suspension of strain XXVIII. This had been titrated to kill all sheep at a dilution of 1:150,000 of a medium sized agar slant (1·5 × 7 cm.) and this dose was much more than an M.L.D. Controls were included with all the immunized sheep. The latter received from 10 to 1,000 times the dose given to the controls. The dilution 1:150,000 has been called a Certain Killing Dose (C.K.D.). The stability of the test suspension was unusually good and strain XXVIII has been the only strain encountered here which retained its virulence in glycerine-saline over long periods. Dilutions of the same suspension were used in all the sheep tests.

All the guinea-pigs which survived in Tables I-V were tested for immunity, but, for the sake of brevity, only the tests with a bearing on the sheep experiments have been given in the following tables. The guinea-pig immunity tests, however, gave no indication that there was any relation between the immunizing power of a strain and the CO_2 concentration in which it developed.

TABLE VI.

Guinea Pigs.	Immunized with.	Tested 3 weeks later with.	Death : Hours after inoculation.						
			20	40	60	80	100	40	80
5	rough avirulent XXII A_2	¼ slant II Ad.	†	†	†				/ †(12 days)
5	Controls	ditto		† † † † †					

Strain II Ad. had not been exalted for this experiment.

TABLE VI—(*continued*).

Sheep.	Immunized with.	Each tested 3 weeks later with.	Death : Days after inoculation. 1	2	3	4	5	6	7	8	
45,825	1 c.c. $\frac{1}{300}$ agar slant XXII A_2	10 C.K.D. XXVIII		†							
46,056					†						
45,988		„		†							
45,742	„	„									/
46,014	„	„									/
45,965	„	„					†				
45,796	Controls.	„			†						
45,807		„		†							
46,731		1 C.K.D.				†					
45,915		„		†							

Strain XXII A_2 was isolated four months previously. The immunity produced in sheep was not good, although two of the sheep survived this fairly severe test dose.

TABLE VII.

Avirulent strain XXVIII K was obtained after 48 hours in 40 per cent. CO_2 in the experiment noted in Table I.

No. of guinea pigs.	Immunized with. (Each.)	Tested 3 weeks later with.	Death : Hours after inoculation. 10	20	30	40	50	60	70	80	90
3	avirulent R from XXVIII K (40% CO_2)	$\frac{1}{8}$ agar slant II Ad. (each)				††					†
6	Controls	ditto		†† † †		† †					

TABLE VII—(*continued*).

Sheep.	Each immunized with.	Tested 3 weeks later with.	Death : Days after inoculation.												
			1	2	3	4	5	6	7	8	9	10	11	12	
46,699	1 c.c. $\frac{1}{500}$	10 C.K.D.													/
45,868	agar slant	strain			†										
46,113	XXVIII K	XXVIII													/
45,908	,,	,,													/
46,060	,,	,,													/
46,077	,,	,,													/
46,688	1 c.c. $\frac{1}{400}$	,,										†			
45,952	agar slant	,,													/
47,052	strain	,,													/
46,109	XXII A_2	,,													/
45,906	isolated	,,													/
46,019	4 months	,,													/
	previously.														
	Vaccine														
	Batch 5														
45,913	20·0 c.c.	,,													/
46,027	20·0 c.c.	,,													/
46,143	0·1 c.c.	,,													/
45,953	0·1 c.c.	,,				†									
45,945	0·1 c.c.	,,													/
46,991	0·1 c.c.	,,													/
45,938	0·01 c.c.	,,													/
46,106	0·01 c.c.	,,													/
46,083	0·01 c.c.	,,													/
46,053	0·01 c.c.	,,													/

NOTE.—The 0·01 c.c. dose B. 5 is equivalent to 1/300 agar slant.

46,083	Controls	10 C.K.D.									†				
46,690	,,	strain		†											
		XXVIII													
		1 C.K.D.													
45,997	,,	,,						†							
47,007	,,	,,				†									

There was only a slight indication of immunity in the guinea-pigs to the large test dose given them.

The same strain tested on sheep however showed a relatively sound degree of immunity (XXVIII K). The immunity in sheep was compared with that produced by an avirulent rough' strain (XXII A_2) isolated some months previously and with that produced by an ordinary vaccine batch. Thus, this relatively poor avirulent strain gave satisfactory immunity when tested on sheep. Controls all died.

TABLE VIII.

Avirulent strain XXXIII B was obtained after 24 hours in 10 per cent. CO_2 and avirulent strain XXXIV F after 24 hours in 30 per cent. CO_2 (see Tables II and IV).

No. of guinea pigs.	Immunized with.	Tested 3 weeks later with. (Each.)	Death : Hours after inoculation. 20	40	60	80	100	60	200
3	XXXIII B (24 hrs.) (10% CO_2)	$\frac{1}{8}$ agar slant II Ad.	†					††	
3	XXXIV F 24 hrs. (30% CO_2)	ditto	† 	 †		 †			
6	Controls	ditto	† † † † † †						

Sheep.	Each immunized with.	Each tested with 3 weeks later.	Death : Days after inoculation. 1	2	3	4	5	6	7	8	
47,014	$\frac{1}{350}$ agar slant	100 C.K.D. XXVIII									/
47,045	R variant				†						/
46,987	XXXIII B				†						
45,963		,,									/
46,692		,,									/
46,054	,,	,,	This sheep was very poor and had pneumonia for a week. Blood and spleen smears negative.							†	
46,748	ditto	,,									/
46,121	XXXIV F	,,									/
46,028	,,	,,									/
47,016	,,	,,		†							
46,726	,,	,,									/
46,756	,,	,,									/
	Batch 6										
46,984	20·0 c.c.	,,									/
44,677	20·0 c.c.	,,									/
45,964	0·1 c.c.	,,									/
45,935	0·1 c.c.	,,									/
46,994	0·1 c.c.	,,									/
47,012	0·1 c.c.	,,									/
45,980	0·01 c.c.	,,									/
46,998	0·01 c.c.	,,									/
46,981	0·01 c.c.	,,									/
46,996	0·01 c.c.	,,									/
	Controls										
45,931	,,	,,			†						
46,051	,,	,,		†							
		1 C.K.D.									
45,852	,,	,,		†							
46,136	,,	,,		†							

The two avirulent strains tested above showed only a moderate degree of protection in guinea-pigs, but a very satisfactory protection in sheep.

TABLE IX.

Avirulent strain XXIV D was obtained after 24 hours in 20 per cent. CO_2 (see Table III).

Guinea pigs.	Each immunized with.	Each tested with.	Death : Hours after inoculation.				
			20	40	60	80	100
3	$\frac{1}{5}$ slant XXIV D	$\frac{1}{4}$ slant II Ad.				†	†
6	Controls	.	† † † †	 † †			

Sheep.	Each immunized with.	Each tested with.	Death : Days after inoculation.							
			1	2	3	4	5	6	7	8
45,871	$\frac{1}{100}$ slant	100 C.K.D.								
45,902	sporulated	XXVIII								
45,886	culture	,,								
46,725	XXIV D	,,								
45,909	,,	,,								
45,944	,,	,,								
45,859	,,	,,								
46,740	,,	,,								
45,971	,,	,,								
46,072	,,	,,								
46,110	,,	,,								
45,922	,,	,,								
Controls		1 C.K.D.								
45,876		,,			†					
45,885		,,		†						
46,038		,,		†						

This avirulent strain produced a high degree of resistance in guinea-pigs and immunized sheep solidly against a large test dose of virulent culture. Sheep were not considered "solidly" immune unless all survived and none showed more than a slight transient temperature reaction to the virulent dose. The majority showed no reaction at all.

TABLE X.

Avirulent strain XXXIV G was obtained after 24 hours in 30 per cent. CO_2; XXXIV M after 48 hours in 75 per cent. CO_2; XXXV A after 48 hours in 10 per cent. CO_2; XXXV B after 48 hours in 20 per cent. CO_2 (see Tables IV and V).

No. of guinea pigs.	Immunized with.	Each tested with.	Death : Hours after inoculation.				
			20	40	60	80	
3	XXXIV G rough 24 hours 30% CO_2	$\frac{1}{8}$ slant II Ad.					/ / /
3	XXXIV M rough 48 hours 75% CO_2	,,			†		/ /
2	XXXV A rough 48 hours 10% CO_2	,,	†	†			
3	XXXV B rough 48 hours 20% CO_2	,,	†††				
12	Controls	,,	† ††† ††† †††	† †			

Sheep.	Each immunized with.	Tested 3 weeks later with.	Death : Days after infection.								
			1	2	3	4	5	6	7	8	
46,075	1 c.c. $\frac{1}{400}$ agar slant XXXIV G	1,000 C.K.D. strain XXVIII									/
45,847											/
45,878											/
45,846	,,	,,									/
46,114	,,	,,									/
46,024	,,	,,									/
46,673	1 c.c. $\frac{1}{400}$ agar slant XXXIV M										/
45,922		,,									/
46,089		,,		†							
46,055	,,	,,									/
46,671	,,	,,									/
45,854	,,	,,		†							
45,883	1 c.c. $\frac{1}{400}$ agar slant XXXV A	,,						†			
45,960		,,				†					
46,091		,,					†				
46,714	,,	,,		†							
45,896	,,	,,									/
46,095	,,	,,				†					

TABLE X—(*continued*).

Sheep.	Each immunized with.	Tested 3 weeks later with.	Death : Days after infection.								
			1	2	3	4	5	6	7	8	
46,015	1 c.c. $\frac{1}{500}$	1,000 C.K.D.									/
46,757	slant	strain									/
46,008	XXXV B	XXVIII									/
46,094	„	„			†						
46,044	„	„									/
45,983	„	„		†							
45,855	1 c.c. $\frac{1}{300}$	„									/
46,040	slant	„									/
46,058	batch 7	„									/
46,741	„	„									/
46,142	„	„									/
46,010	„	„									/
46,765	1 c.c. 1/30	„									/
46,138	slant rough	„									/
45,851	avirulent	„									/
46,724	XXII A_2	„									/
46,764	„	„									/
46,674	„	„									/
	Controls										
46,105		10 C.K.D.		†							
46,747		„		†							
45,873		1 C.K.D.			†						
45,863		„			†						

In this experiment, two avirulent rough strains which gave very good immunity in guinea-pigs and two avirulent rough strains which gave very poor immunity in guinea-pigs were compared with a rough avirulent strain ten times as concentrated, and an ordinary vaccine (Pasteur II) type. The concentrated suspension of the avirulent rough strain XXII A_2 was prepared four month previously from an avirulent strain isolated 8 months previously. (See Tables VI and VII for tests on this strain at $\frac{1}{300}$ dilution.) Four uninoculated controls were included.

Strain XXXIV G immunized guinea-pigs and sheep solidly. XXXIV M increased the resistance of guinea-pigs considerably but was only moderately effective in sheep. Strain XXXV A gave a poor result in both guinea-pigs and sheep. XXXV B gave a poor result in guinea-pigs and immunized the sheep fairly well.

The concentrated strain XXII A_2 and the Pasteur type vaccine batch 7 both produced a solid immunity in sheep.

It should be noted that the test dose used in this experiment was particularly severe.

DISCUSSION.

Most of the avirulent variants produced a high degree of resistance in sheep and two of them, as judged by the tests, gave a solid immunity. The successful variants were those that showed the highest protective power in guinea-pigs. Avirulent dissociants can be isolated easily and rapidly, so that for practical purposes only those giving sound results in guinea-pigs need be tried on sheep. Nevertheless, even strains which produced a relatively poor immunity in guinea-pigs were comparatively successful when tried on sheep.

However, considerable variation existed between the degrees of immunity produced by the different strains. The reason is unknown but the conditions under which the variants can be obtained are as yet ill-defined and the problem may be solved by a refinement in technique and a more rigid standardisation of the medium. This is the more likely because differences in growth were noted with different batches of media and sera.

The immunity produced by the avirulent dissociants was compared with that given by two batches of an ordinary vaccine strain (Pasteur II type). This gave as good an immunity as the best of the roughs. However, this Pasteur type vaccine was exceptional in that it elicited a better immunity than any similar vaccine prepared here for some time. Moreover, its virulence was on the borderline of safety. The experiments were slightly weighted against the avirulent strains as far as dosage was concerned.

The experiment with strain XXII A_2 noted in Table X introduced an interesting possibility. The usual dose of this strain did not produce a strong immunity in guinea-pigs or sheep, but when ten times this concentration was given the sheep acquired a solid immunity, although the glycerine-saline suspension of strain XXII A_2 was then four months old.

This tenfold strength contained about 3,000,000 spores per dose and it is quite feasible to manufacture vaccines containing this number of organisms. Therefore, it appears probable that larger doses of potent avirulent strains may give maximal immunity to anthrax. It has been shown that large doses of the ordinary vaccines improve immunity, but this increased dosage carries with it an increased risk, whereas almost any dose of the avirulent strains can be tolerated.

The immunity tests with the avirulent rough variants must be considered very satisfactory and indicate, as far as laboratory experiments can, that domestic animals can be easily and safely immunized against anthrax with avirulent strains. This is particularly important for goats, horses and wild animals in zoological gardens (Neitz, 1936). These animals are highly susceptible to the use of any but weak, poorly immunizing Pasteur I type vaccines, which means that these animals are practically non-immunizable. Potent avirulent strains should prove very useful in such cases. In laboratory tests not quoted here, one C.K.D. of strain XXVIII killed almost every goat treated with the Pasteur I type of vaccine, whereas it was shown (Sterne, 1937) that a weak avirulent strain produced fair immunity in goats. The terms Pasteur I and Pasteur II type vaccines refer only to the relative virulence of the attenuated strains.

SUMMARY.

(1) Virulent anthrax strains grew mucoid and produced avirulent rough dissociants on serum agar, in a number of different carbon dioxide concentrations.

(2) A relatively small dose of avirulent spores immunized sheep against anthrax.

(3) The immunizing power of the avirulent rough strains was not associated with the carbon dioxide concentration in which they developed.

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Section IV.

Parasitology.

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Some Undescribed Species of the Nematode Genus Physaloptera Rud., together with a Key to the Sufficiently Known Forms.

By R. J. ORTLEPP, Section of Parasitology, Onderstepoort.

THE species discussed in the ensuing pages, except *P. dispar* von Linst., 1904, comprise some of the material which has from time to time been sent to this institute for determination. As this genus comprises a considerable number of species, and as the writer has found the accompanying key to be of considerable help in his determinations, he has thought it advisable to place this key at the disposal of other workers, and at the same time give a description ot those new forms which he has been able to study.

A paper by Schultz (1927b), which appears to be very comprehensive, deals with the fam. Physalopteridae and the principles underlying the classification of its members. Unfortunately, the writer has not been able to master the contents of the Russian text and he therefore does not think he would be doing justice to Schultz's paper by passing any comments based solely on the brief German summary.

The key only deals with those species of which sufficient data is available for their inclusion. Unfortunately the recently described species *P. achari* Mirza, 1935, *P. leiperi* Skrj., 1924, and *P. seurati* Issaitchikov, 1926, had to be omitted as no descriptions of these were available. The subspecies have also been omitted.

PHYSALOPTERA DISPAR von Linstow, 1904.

syn P. CLAUSA Rud, 1819 of Seurat, 1917.

not P. CLAUSA Rud, 1819.

In a previous communication (1922) the writer expressed the view that the material described by Seurat (1917) as *P. clausa* Rud. was not the same as the material on which Rudolphi had based his description, which material the writer had examined, and that it probably represented a new species. Seurat considered that his specimens were the same as *P. dispar* v. Linst., which species he listed as a synonym of *P. clausa* Rud. The writer, however, expressed the view that until the types of von Linstow's species were re-examined, Seurat's and von Linstow's materials should not be regarded as co-specific. Baylis (1928) has fortunately been able to re-examine von Linstow's type material from *Erinaceus albiventris* which are in the British Museum, London, and he found that this material agreed " in every important respect with the description of '*P. clausa*' given by Seurat (1917) ", and that von Linstow's description was inaccurate in several respects; further that the length of the left spicule was more in accord with von Linstow's findings than with Seurat's. In consequence of Baylis' observations the identity of Seurat's material is now certain and it does not represent a new species as the writer thought.

Stomach: *Atelerix spiculus, A. spinifex, A. hindei sotikae, Erinaccus algirus, E. deserti* and *E. albiventris*, North and Central Africa.

PHYSALOPTERA IMMERPANI sp. nov.

The material on which the description of this species is based was recovered from the stomachs of two hedgehogs* obtained one from Mr. J. Todd's farm at Immerpan and the other from Mr. R. V. Mitchell's farm Pienaar's River; both these farms are in the Northern Transvaal and are about 100 miles distant from each other. Each hedgehog was parasitised by about a dozen worms, all of which were the same. The parasites, which were firmly attached to the stomach, were recovered soon after death of the hosts, and they were killed and fixed in warm 70 per cent. alcohol.

The cuticle is finely striated and is partially or wholly reflected over the lips. The cervical papillae are symmetrically placed and occupy a varying position from the level of the junction of the two oesophageal parts to about 0·2 mm. behind this level: the excretory pore follows the position of the papillae and is found just posterior to them.

The lips are hemispherical and each carries a dome-shaped papilla towards each of its latero-dorsal and latero-ventral borders. At its apex there is a massive and well chitinized external tooth having a rounded tip, and internal to it there is a tripartite tooth of the same size. No other teeth are present. The oesophagus is straight and becomes thickened posteriorly; it consists of the usual two parts, muscular and glandular. In the larger females it is from 5·6 to 7·75 mm. long and occupies from one-seventh to one-sixth of the body length: its muscular portion is from 0·56 to 0·78 mm. long, forming about one-tenth of the whole organ. In the larger males the oesophagus is from 5·1 to 5·3 mm. long and occupies from one-fifth to two-ninths of the body length; its muscular portion varies in length from 0·52 to 0·58 mm. forming from one-tenth to one-ninth of the total length of the oesophagus. The nerve ring encircles the oesophagus in its posterior quarter.

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* The writer wishes to express his sincere thanks to Mr. W. O. Neitz, B.V.Sc., for having placed these carcasses at the writer's disposal.

Female.—The females are large and robust and reach a length of 53 mm. and thickness of 1·4 mm.; they are thickest in their middle, and from this level the body becomes attenuated anteriorly, and slightly less so posteriorly. The tail is relatively short, stumpy and pointed and varies in length from 0·26 to 0·3 mm. In ten females examined the vulva was situated anterior to the end of the oesophagus in all; the distance varied, however, from 0·5 to 2·5 mm.; it is non-protuberant. The vagina passes straight back and is from 2 to 2·5 mm. long; its outer outline is slightly corrugated, and its thickness averages about 0·07 mm. The egg chamber, which is somewhat spindle-shaped, is from 1·3 to 1·5 mm. long with a maximum thickness of 0·34 mm.; it contains very few eggs, even in specimens whose uteri are completely filled and expanded by mature eggs. The two uteri take their origin immediately posterior of the egg-chamber, a trunk portion being absent; in some specimens, however, the origins of the uteri may be closely opposed and so give a false impression of a trunk. The eggs are small, oval and embryonated *in utero*; their shell is about 0·003 mm. thick. Their size is 0·055 to 0·061 mm. long by 0·038 to 0·041 mm. broad.

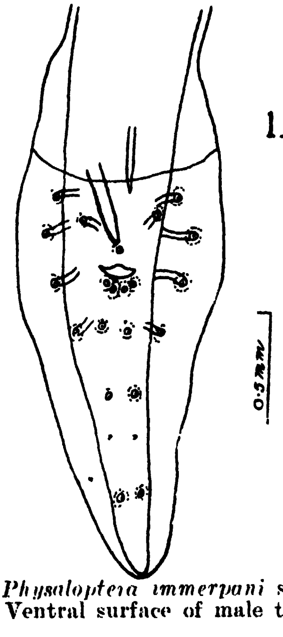


Fig. 1. *Physaloptera immerpani* sp. n.
Ventral surface of male tail.

Male.—The males are considerably smaller and thinner than the females, and may reach a length of 25 mm. with a maximum thickness of 0·58 mm. just in front of the caudal expansions. Anteriorly the body tapers, but posteriorly except for the tail region, this is not marked. The tail is bent ventrally and is from 1 to 1·2 mm. long; its ventral surface is covered for the most part by longitudinal rows of tubercles. When flattened out the caudal expansion gives the posterior extremity a spear-head shape supported by the pedunculated lateral papillae; the cuticle in front of the papillae is inflated ventrally giving this portion a bulbous shape in side view. The arrangement of the caudal papillae (Fig. 1) is very similar to that found in *Ph. dispar* and *Ph. tacapensis,* the only difference being that the third pair of post-cloacal ventral papillae is sessile and not stalked.

The spicules are small and subequal, that of the left being the more slender and tapers to a fine point; it is from 0·23 to 0·33 mm. long; the right spicule is robuster, has a rounded tip, and is similar in shape to that found in *Ph. dispar* and *P. tacapensis;* it is from 0·29 to 0·32 mm. long; it is sometimes longer and sometimes shorter than the left.

Host: Atelerix frontalis.

Habitat: Stomach.

Occurrence: Northern Transvaal.

Types in helminthological collection, Onderstepoort.

Affinities.—Ph. immerpani, together with *Ph. dispar* and *Ph. tacapensis* form a very closely inter-related group; they all have two uteri, originating directly from the base of the egg-chamber; short spicules of which the right is somewhat thimble-shaped; the caudal ornamentation in the male is very similar and the lips are provided with the same type of teeth. In *Ph. immerpani* and *Ph. tacapensis* the vulva is in front of the end of the oesophagus, whereas it is post oesophageal in *Ph. dispar.* The above described species can be differentiated from *Ph. tacapensis* by its much larger size (53 mm. as against 22 mm. in female), much thinner egg-shell (0·003 against 0·007 mm.); longer spicules and sessile nature of the third pair of ventral papillae.

Von Linstow (1908) described a new species—*Ph. incurva* from the same host obtained from the Kalahari; apart from the lips, which von Linstow states are dorsal and ventral, his species differs from the writer's in that the vulva is post oesophageal and the spicules are much longer (right 0·36 and left 0·57 mm.).

Specific Diagnosis.—Fairly large worms, females up to 53 mm. and males up to 25 mm. long. External tooth massive and with blunt tip; internal tooth of same height, with tripartite tip. Vulva in oesophageal region; two uteri; trunk portion absent; caudal papillae of male as in *Ph. dispar,* except that third ventral pair is sessile. Spicules subequal, the left sometimes longer, sometimes shorter than the right; left spicules 0·23 to 0·33 mm. long, right spicule 0·29 to 0·32 mm. long and somewhat thimble-shaped.

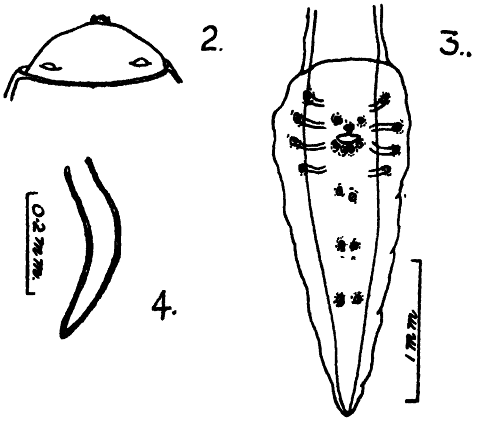
Stomach, *Atelerix frontalis,* Northern Transvaal.

PHYSALOPTERA LOSSENI sp. nov.

The material consisted of three adult females and one adult male; in addition the posterior body half of another male was also present. The material had been collected from a hawk by Mr. R. Lossen, Ongeama, S.W. Africa and was submitted to this institute for identification. Mr. Lossen stated that about 25 specimens were collected from the pharynx.

The body is fairly robust and is covered by a finely annulated cuticle which is partially reflexed over the lips. The cervical papillae are symmetrically placed, small and spike-like and are found from 0·2 to 0·28 mm. behind the posterior end of the muscular oesophagus; the excretory pore occupies the usual position, just posterior to the cervical papillae.

The two lateral lips are dome-shaped and each carries two prominent papillae towards their dorsal and ventral margins (Fig. 2). The external tooth is large and cone-shaped and the inner membranous tripartite tooth is also large and only slightly shorter than the outer; lateral teeth and denticles are absent. In the females the oesophagus is from 4·2 to 4·3 mm. long and in the single male it is 3·7 mm. long. In the former the muscular portion is from 0·57 to 0·62 mm. long and in the male 0·45 mm. The nerve ring encircles this portion at about the junction of its second and last thirds.



Figs. 2–4. *Physaloptera losseni* sp. n.
,, 2. Lateral view of lip.
,, 3. Ventral view of male tail.
,, 4. Right spicule.

The three females are respectively 29, 31 and 32 mm. long with thickness of 1 to 1·3 mm. They are attenuated towards both extremities and the body is terminated by a pointed tail 0·6 to 0·62 mm. in length. The vulva is non-protuberant and is situated just posterior to the hind end of the oesophagus; it leads into a vagina 2·3 mm. long by 0·08 mm. thick; the following egg-chamber is just slightly shorter (2·1 mm.) and had a maximum diameter of 0·4 mm.; the trunk is about 0·75 mm. long by 0·09 mm. thick and gives rise to the two uteri. The eggs, which were confined to the uteri, were oval, smooth, thick-shelled and embryonated; they were from 0·047 to 0·049 mm. long by 0·035 to 0·037 mm. broad.

The solitary complete male was 24 mm. long with a maximum thickness of 0·84 mm. just anterior to the caudal expansions. Anteriorly the body becomes slightly attenuated. The tail is ventrally flexed and is 1·9 mm. long. When opened out the caudal expansions give the tail a lanceolate shape (Fig. 3). The four circum-cloacal stalked papillae are situated two pre- and two post-cloacal, and the centre two have longer stalks. The three pre-cloacal ventral papillae form a flattened triangle and the central papilla is the largest. The 1st and 2nd pairs post-cloacal ventral papillae are small and situated in a transverse row on the posterior lip of the cloaca; the distances between these and the 3rd, 4th and 5th pairs are about equal and these three pairs are found at the posterior limits of the 1st, 2nd and 3rd fifth of the tail respectively; in one tail the 5th ventral papilla on the right is shifted forwards and lies adjacent to the 4th papilla, forming a single double papilla. The spicules are very unequal but unfortunately all, except one right spicule, are broken; the entire right spicule is boomerang-shaped and pointed, 0·4 mm. long and 0·058 mm. thick (Fig. 4); the remaining portions of the left spicules are respectively 1·86 and 3·2 mm. long and 0·046 and 0·05 mm. thick; they are filiform but their tips have unfortunately broken off. The greater portion of the central area of the tail is ornamented with longitudinal rows of tubercles typical for the genus.

Host: Spizaetus bellicosus.

Habitat: Pharynx.

Occurrence: South West Africa.

Types in the helminthological collection, Onderstepoort.

Affinities.—The tripartite nature of the inner tooth and mode of origin of the two uteri allies this species to *P. maxillaris*, from which species it may, however, be differentiated by the boomerang-shape of its right spicule, its much longer left spicule, the position of its ventral pre-cloacal papillae, and by the difference in cuticular ornamentation on the ventral surface of its tail.

Specific Diagnosis.—Robust forms up to 32 mm. long, with large conical outer and tripartite inner teeth. Two uteri arising from a common trunk; vulva post-oesophageal. Spicules very unequal, right boomerang-shaped and less than 0·5 mm. long; left long and filiform more than 3 mm. long.

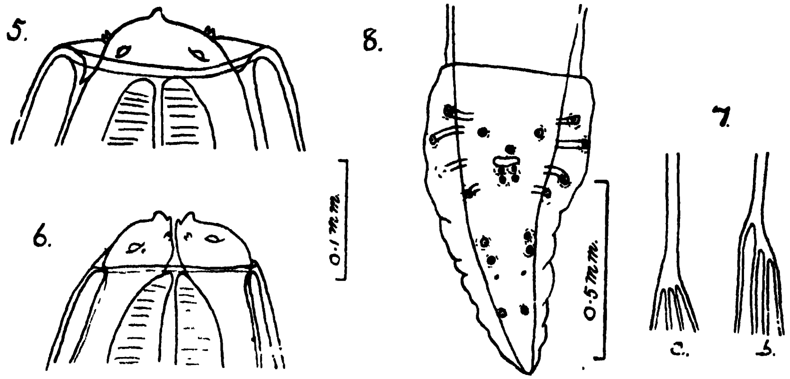
. Pharynx, *Spizaetus bellicosus*, S. W. Africa.

Physaloptera tasmani sp. nv.

This material consisted of about a dozen males and females collected by the Rev. Tasman from a chameleon at Kutuma, Southern Rhodesia. Unfortunately the specimens are all adolescent, no females possessing eggs and some males and females were still unsheathed in the cuticle of the previous stage. These specimens not being mature the writer is not giving a detailed description but is confining himself to the salient features only.

The largest specimens are only about 15 mm. long and the cuticle is only slightly reflected over the lips. The position of the cervical papillae, excretory pore and nerve ring are typical for the genus. The two lateral lips (Figs. 5 and 6) are conical and rounded and each carries two dome-shaped papillae, a prominent triangular external tooth, a small spike-like inner tooth and two bifid lateral teeth; between these teeth there is a single row of small denticles.

The vulva has a post-oesophageal position, just behind the oesophagus; the vagina is about 1 mm. long and 0·035 mm. thick and leads into a poorly developed egg chamber about 0·3 mm. long by 0·09 mm. in diameter. A relatively long trunk portion follows (0·4 mm.) and gives rise to the four uteri at the same level as in *Ph. paradoxa;* in one of four females in which this origin could be clearly followed, one uterus originated slightly more anterior to the remaining three (Figs. 7a and b).



Figs. 5–8. *Physaloptera tasmani* sp. n.
,, 5. Lateral view of anterior extremity.
,, 6. Ventral view of anterior extremity.
,, 7. Variations in mode of origin of uteri.
,, 8. Ventral view of male tail.

In the males the caudal expansions are present although not very prominent, and the outline of the tail is spear-shaped (Fig. 8). The arrangement of the papillae are as seen in *P. paradoxa*, except that the 3rd and 4th post-cloacal ventral pairs are not adjacent to each other. The spicules are only weakly chitinized and appear to be only slightly unequal. The right is about 0·178 to 0·2 mm. long, and is somewhat spear-shaped; the left is slender and pointed and appears to be about 0·2 mm. long.

Host: Chamaeleon macrolepis.

Habitat: Stomach.

Occurrence: South Rhodesia.

Types in helminthological collection, Onderstepoort.

Affinities.—This species appears to be closely related to *Ph. paradoxa* von Linstow, described from *Varanus albigularis* and various snakes; it differs from it, however, in that the denticles do not extend laterally beyond the lateral teeth; the position of the third and fourth post-cloacal ventral papillae is different and in the apparently much shorter left spicule.

Two species of Physaloptera have been recorded from chamaeleons namely *Ph. leptosoma* (Gervais, 1848) Seurat, 1917 (syn. *Ph. chamaeleontis* Gedoelst, 1916) and *Ph: ortleppi* Sandground, 1928. Gedoelst's material originated from *Chamaeleon gracilis*, Belgian Congo, while Sandground obtained his from *Chamaeleon dilepis*, Tanganyika. The former species can be excluded from comparison in that it is didelphic, but Sandground's species appears to be closely related; this species may, however, be distinguished from the writer's by the absence of lateral teeth, the dichotomous branching of its uteri, and the great unequality of the two spicular lengths.

Specific Diagnosis.—Tetradelphic forms, with the uteri arising at the same level; lips provided with external triangular and lateral bifid teeth; between these a row of small denticles; internal tooth small and spike-like. Vulva post-oesophageal. Spicules differ apparently only slightly in length, left slender and pointed, right robuster and spear-shaped; and 0·2 mm. and 0·178 to 0·2 mm. long respectively.

Stomach, *Chamaeleon macrolepis*, South Rhodesia.

KEY TO SPECIES OF PHYSALOPTERA.

1.	Two uteri present (Group **Didelphys**)............	—	
	Three uteri present (Group **Tridelphys**)..........	**P. cebi** Ortlepp, 1923.	
	Four uteri present (Group **Tetradelphys**).........	—	40
	More than four uteri present (Group **Polydelphys**)	—	61

GROUP DIDELPHYS.

2.	Uteri arise direct base of egg chamber...........	—	3
	Uteri connected to egg chamber by a common trunk	—	16
	Mode of origin of uteri not definitely stated......	—	36
3.	Uteri arise from sides of egg chamber like two horns	—	4
	Uteri arise close together from base of egg chamber	—	9
4.	Cuticle reflexed over tail.......................	—	5
	Cuticle not reflexed over tail...................	—	7
5.	Ventral surface of male tail ridged.............	—	6
	Ventral surface of male tail rugose	**P. praeputialis** v. Linstow, 1889.	
6.	External and internal teeth of same height......	**P. malayensis** Ortlepp, 1922.	
	External tooth shorter than internal tooth......	**P. canis** Mönnig, 1929.	
7.	Lateral teeth on shoulder of lips ; female only known	**P. quadridentata** Walton, 1927.	
	No lateral teeth on shoulder of lips..............	—	8
8.	Left spicule over 1 m.m. long..................	**P. acuticauda** Mol., 1860.	
	Left spicule less than 1 m.m. long. Vulva near middle of body	**P. terdentata** Mol., 1860.	

9.	Vulva near middle of body: female only known	**P. brevivaginata** Seurat, 1917.	
	Vulva towards anterior part of body	—	10
10.	Tip of left spicule spear-shaped	**P. bonnei** Ortlepp, 1922.	
	Tip of left spicule pointed	—	11
11.	Right spicule robust and somewhat thimble-shaped	—	12
	Right spicule slenderer and pointed	—	14
12.	Vulva post-oesophageal	**P. dispar** v. Linst., 1904.	
	Vulva in oesophageal region	—	13
13.	Third pair ventral post-cloacal papillae stalked	**P. tacapensis** Seurat, 1917.	
	Third pair ventral post-cloacal papillae sessile	**P. immerpani** sp. n.	
14.	Vulva generally in oesophageal region	**P. getula** Seurat, 1917.	
	Vulva post oesophageal	—	15
15.	No denticles on lips	**P. clausa** Rud. 1819.	
	Traces of denticles on lips	**P. mydai** Baylis, 1926.	
16.	Muscular and glandular oesophageal parts equal	**P. rara** Hall & Wigdor, 1918.	
	Muscular portion much shorter	—	17
17.	Lateral papillae all precloacal and in two widely separated groups	**P. longissima** Ortlepp, 1922.	
	Lateral papillae not all pre-cloacal and not in two widely separated groups	—	18
18.	Left spicule more than 1 m.m. long	—	19
	Left spicule less than 1 m.m. long	—	21
19.	Lateral teeth on lips present	**P. leptosoma** (Gerv., 1848).	
	Lateral teeth on lips absent	—	20
20.	Left spicule less than 2 m.m. long	**P. maxillaris** Mol., 1860.	
	Left spicule greater than 2 m.m. long	**P. losseni** sp. n.	
21.	Left spicule over 0·5 m.m. long	—	22
	Left spicule less than 0·5 m.m. long	—	23
22.	Vulva at level of end of oesophagus	**P. semilanceolata** Mol., 1860.	
	Vulva post-oesophageal	**P. subalata** Sch., 1866.	
23.	Tip of left spicule spear-shaped	—	24
	Tip of left spicule not spear-shaped	—	25
24.	Vulva at level of end of oesophagus	**P. monodens** Mol., 1860.	
	Vulva post-oesophageal	**P. obtuissima** Mol., 1860.	
25.	Rugosity on male tail on cloacal pad only	**P. gracilis** Ortlepp, 1922.	
	Rugosity on male tail not on cloacal pad only	—	26
26.	Right spicule hooked or slightly bent	**P. retusa** Rud., 1918.	
	Right spicule not hooked or slightly bent	—	27
27.	Single tooth (external) on each lip	**P. phrynosoma** Ortlepp, 1922.	
	Two or more teeth on each lip	—	28

28.	External and internal teeth small	**P. muris-brasiliensis** Dies., 1861.	
	External and internal teeth large	—	29
29.	Teeth equal or subequal	—	30
	Teeth unequal	—	31
30.	Cuticle inflated: Spicules feebly chitinized	**P. bedfordi** Ortlepp, 1932.	
	Cuticle not inflated; spicules well chitinized	**P. anomala** Mol., 1860.	
31.	External tooth smaller than internal tooth	—	32
	External tooth larger than internal tooth	—	34
32.	Vulva near middle of body	**P. galinieri** Seurat, 1914.	
	Vulva towards anterior end; 5 pairs lateral papillae.	—	33
33.	Five pairs ventral postcloacal papillae	**P. rapacis** Mönnig, 1926.	
	Six pairs ventral postcloacal papillae	**P. reevisi** Chu, 1931	
34.	Large forms up to 68 m.m. long from Edentates	**P. papillotruncata** Mol., 1860.	
	Smaller forms from hawks.	—	35
35.	Unpaired papilla between last pair of ventral caudal papillae	**P. crossi** Seurat, 1914.	
	Unpaired papilla absent	**P. alata** Rud., 1819.	
36.	Vulva in posterior body half	**P. bispiculata** Vaz & Pereira, 1935.	
	Vulva in anterior body half	—	37
37.	Caudal alae and papillae reduced; spicules feebly chitinized	**P. torquata** Leidy, 1886.	
	Caudal alae and papillae not reduced; left spicule chitinized	—	38
38.	Only one ventral caudal papilla	**P. spinicauda** McLeod, 1933.	
	Several ventral caudal papillae	—	39
39.	Left spicule less than 0·5 m.m. long	**P. squamatae** Harwood, 1932.	
	Left spicule greater than 0·5 m.m. long	**P. cerdocyona** Sprehn, 1932.	

GROUP TETRADELPHYS.

40.	Single tooth (external) only on each lip	—	41
	Lips carry more than one tooth	—	42
41.	Small forms up to 8 m.m. long	**P. colubri** (Rud., 1819)...	
	Large forms up to 44 m.m. long	**P. simplicidens** Ortlepp, 1922.	
42.	Male unknown	—	43
	Male known	—	44
43.	From Gerbil, N. Africa	**P. numidica** Seurat, 1917.	
	From Australian reptiles	**P. clelandi** Irwin-Smith, 1922.	
44.	Left spicule over 3 m.m. long	—	45
	Left spicule less than 3 m.m. long	—	46

45. Left spicule over 4 m.m. long.................. **P. caucascia** v. Linst., 1907.
Left spicule less than 4 m.m. long.............. **P. africana** (Mönnig, 1924).

46. Left spicule over 1 m.m. long.................. — 47
Left spicule less than 1 m.m. long.............. — 55

47. Uteri do not arise by dichotomous branching.... **P. paradoxa** v. Linst., 1908.
Uteri arise by dichotomous branching........... — 48

48. Tip of left spicule spear-shaped.................. **P. antarctica** v. Linst., 1899.
Tip of left spicule pointed...................... — 49

49. Denticles absent.............................. — 50
Denticles present.............................. — 51

50. Tip of right spicule bent and blunt............. **P. vandenbrandeni** Gedoelst, 1924.
Tip of right spicule straight and sharp.......... **P. varani** Parona, 1889.

51. Inner tooth absent or indistinct................ — 52
Inner tooth present........................... — 53

52. Vulva near middle of body...................... **P. physignathi** Baylis, 1924.
Vulva in anterior body third.................. **P. ortleppi** Sandground, 1928.

53. Denticles on lower angles of lips only........... **P. bancrofti** Irwin-Smith, 1922.
Denticles on anterior margin of lips............ — 54

54. Dichotomous branching of uteri very short...... **P. quadrovaria** Leiper, 1908.
Dichotomous branching of uteri fairly long...... **P. abbreviata** Rud., 1819.

55. Uteri arise directly from base of egg chamber... **P. tumefaciens**, Henry & Blanc, 1912.
Uteri arise by branching of common trunk...... — 56

56. Denticles absent.............................. — 57
Denticles present.............................. — 58

57. Inner tooth large and tripartite................ **P. magnipapilla** Mol., 1860.
Inner tooth small and spike-like................ **P. leidyi** Walton, 1927.

58. Vulva in oesophageal region.................... **P. pallaryi** Seurat, 1917.
Vulva post-oesophageal........................ — 59

59. Inner tooth absent........................... **P. amaniensis** Sandgr., 1928.
Inner tooth present........................... — 60

60. Uteri arise by dichotomous branching of common trunk **P. polydentata** Walton, 1932.
Uteri do not arise by dichotomous branching of common trunk **P. tasmani** sp. n.

GROUP POLYDELPHYS.

61.	Five uteri present	**P. musculi** Thwaite, 1927.	
	More than 5 uteri present	—	62
62.	Six to 7 uteri	—	63
	Number of uteri more than 7	—	64
63.	Very large forms with lateral teeth (from Suidae).	**P. aduensis** Baylis, 1928.	
	Smaller forms without lateral teeth (from Rodents)	**P. joyeuxia** Gendre, 1928.	
64.	Uteri arise by dichotomous branching	**P. capensis** Ortlepp, 1922.	
	Uteri dot not arise by dichotomous branching	—	65
65.	Outer tooth truncated	**P. multi-uteri** Canovan, 1929.	
	Outer tooth conical	—	66
66.	Fourth and fifth post-anal papilla in a transverse row	**P. turgida** Rud., 1819.	
	Fourth and fifth post-anal papilla not in a transverse row	—	67
67.	Third, fourth and fifth post-anal papillae equidistant and in anterior half of tail	**P. torresi** (Trav., 1920).	
	Third, fourth and fifth post-anal papillae not equidistant and in posterior half of tail	**P. dilatata** Rud., 1819.	

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A hitherto unrecorded Filaria, *Suifilaria suis*, N.G., N.Sp., from the Domestic Pig in South Africa.

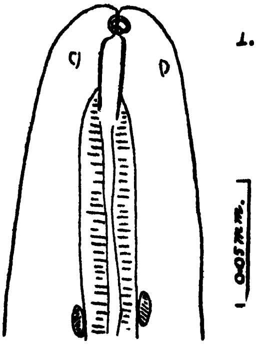
By R. J. ORTLEPP, Section of Parasitology, Onderstepoort.

About a year ago portions of preserved pig muscle were submitted by the Port Elizabeth abattoir authorities to this Institute in order to determine whether some whitish oval cysts attached to the fasciae of the muscles had any relation to cysticercosis. On dissection it proved that these cysts enclosed a delicate coiled worm, and the writer was fortunate in obtaining one complete female together with about 6 anterior and posterior ends of other female worms; no males were recovered. Examination of this material clearly showed the filariid nature of this parasite, and in addition characteristics were noted which did not agree with the description of any filariid known to the writer. The services of the Government Veterinarian at Port Elizabeth were now solicited and Mr. Clemow, B.V.Sc., kindly undertook to obtain further material; in consequence some entire males and females were obtained and sent to this Institute together with a portion of unpreserved pig flank and a portion of flank was sent in glycerine; these showed several cysts under the body cavity lining. From these cysts the writer was able to dissect out several entire males and females. The writer wishes to express his sincere gratitude to Mr. Clemow for his kindly services.

When transmitting the above material Mr. Clemow wrote as follows:—" . . . I saw the usual filaria under the fasciae of the muscles of the fore-quarters, and a number of cysts in the fat under the flank muscles. . . . According to the Meat Inspector the worms are found under the fasciae of any of the muscles in severe cases, but particularly those of the fore-quarters. I find, . . . that they can be detected by skinning the forearm and examining the fasciae ". From Mr. Clemow's communication it appears that these parasites may be either free or encysted; the free parasites sent were mature males and females and those dissected out by the writer from the cysts, were also mature. Whether encystment is normal to the parasite remains undetermined.

The material available for study consisted of about 15 entire males and females, together with several anterior and posterior ends of both sexes. They are thin white worms, the females being about half as long again as the males and slightly thicker; the males are from 17 to 25 mm. long with a maximum thickness of 0·1 to 0·13 mm. and the females are from 32 to 40 mm. long by 0·15 to 0·17 mm. thick in the middle. The cuticle shows no signs of annulations in both sexes, and lips are also absent. The head is ornamented by two lateral cuticular auricular-like structures, which probably represent the modified lateral papillae of the head (Fig. 1): from each a duct passes backwards and joins a pear-shaped gland lying on the side of the oesophagus and anterior to the nerve ring. In addition, just posterior to the level of the auricular-like structures there are 4 very indistinct submedian head papillae which cause only a very slight bulging of the cuticle. The mouth is a small round aperture which leads through a short narrow canal to the small and cylindrical buccal capsule, about 0·025 mm. long and 0·009 mm. broad, and having a cuticular wall about 0·001 mm. thick. The oesophagus is extremely long, and its junction with the intestine was in most cases not determined; it would appear as if it insensibly passed over into the intestine: In those specimens where some indication of its posterior limit was seen, it reached almost to the middle of the body, its ratio to the total length being as 5:6. It consists of a short anterior muscular and a long and thicker posterior glandular portion; the anterior portion was found to be from 0·2 to 0·25 mm. long by 0·02 to 0·026 mm. thick, and it was encircled by the nerve ring at about the junction of its first and second thirds. The excretory pore was not seen.

1.



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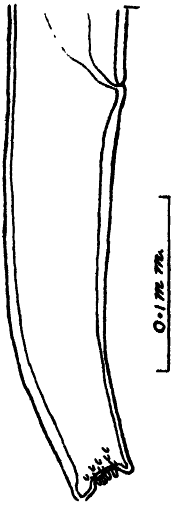

Fig. 1. *Suifilaria suis.* Lateral view of anterior extremity.
Fig. 2. *Suifilaria suis.* Tail of female.

In the female the tail (Fig. 2) is straight and is from 0·186 to 0·226 mm. long; it has the appearance of a truncated cone, the blunt end of which is provided with a number of knobs or tubercles. These are of two kinds, large and small; of the former there are usually three, one dorsal and two ventro-lateral, but this arrangement is liable to vary and one may be ventral and two dorso-lateral. Between these larger tubercles fifteen to twenty smaller tubercles are irregularly scattered about.

The vulva is situated just posterior of the level of the auricular-like head structures and about 0·015 mm. from the anterior end; it leads into a long and straight muscular vagina, 3·4 to 4·3 mm. long by 0·03 mm. wide and having a cuticular lining. Two uteri take their origin from its posterior end, and these pass straight down the length of the body and then bend and pass forwards. Each contains numerous oval, smooth and thin-shelled eggs, and those near the beginning of the vagina are embryonated: These eggs are 0·051 to 0·061 mm. long, 0·028 to 0·032 mm. broad. No free embryos were noted in the vagina, neither were any found in blood smears taken from an infected pig.

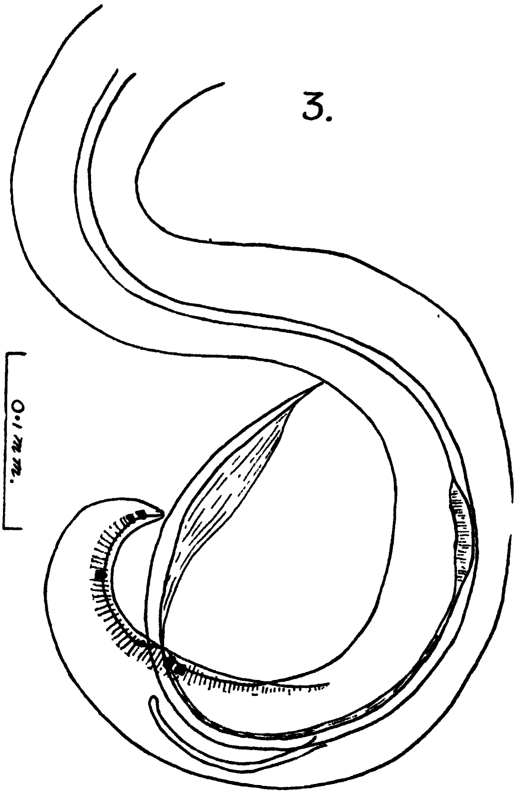

Fig. 3. *Suifilaria suis.* Posterior extremity of male.

In the male the posterior end of the body (Fig. 3) forms two to four loose spirals and is terminated by a conical tail 0·145 to 0·157 mm. long. A narrow caudal ala, about 0·015 mm. wide is present on the left side and extends from about 0·2 mm. anterior of the cloaca to the tip of the tail: It is continued round the tail tip on to the right side where it extends forwards for about 0·05 mm.; except for this small ala at the tip of the tail, the right side is entirely non-alate. The ala shows coarse transverse markings extending from the body through about two-thirds of its width. The caudal papillae are not very distinct, but the writer was able to make out the following which appear to be typical. There are two pairs of sessile papillae on either side just anterior to and a smaller more ventral pair just posterior of the cloaca. Just posterior of the middle of the tail, there is a further small lateral pair, and at the beginning of the last tail quarter two additional pairs of small papillae are present. On the right side the small alae may terminate either anterior or posterior of these last papillae. The spicules are well cuticularized, dissimilar and unequal; the right, 0·105 to 0·115 mm. long by about 0·005 mm. thick at its base, is curved and terminates in a slightly bulbous tip: The left spicule is stouter, about 0·01 mm. thick at its base and from 0·655 to 0·87 mm. long and terminates in a point. It consists of two more or less equal parts, an anterior non-alate portion and a posterior-alate, the two halves being joined to one another by a thicker and somewhat membranous portion.

This parasite, which is the first record, as far as the writer is aware, of a filaria from the domestic pig in South Africa appears to be not only a new species, but also the representative of a new genus of the Filarinae and the following genus and species is created for its reception:

SUIFILARIA SUIS N.G., N. SP.

Generic and Specific Diagnosis.—Filariinae; mouth simple, without lips; lateral papillae modified to form cuticular auricular-like structures; small buccal capsule; oesophagus very long, consisting of a short anterior muscular portion and long posterior glandular portion. Vulva near mouth; long vagina and two uteri; female tail with a number of tubercles at its end. Caudal end of male coiled: Complete ala on left side only; spicules very unequal and dissimilar.

Type species. *S. suis* N. Sp. from *Sus scrofa domestica*, free in fasciae of muscles or encysted.

Affinities.—The simple nature of the mouth, the absence of a peribucca ring, the anterior position of the vulva and the unequal and dissimilar spicules places this parasite in the sub-family Filariinae Stiles, 1907. It differs, however, from all the known genera of this sub-family in the combined presence of a buccal capsule, the modified lateral head papillae to form cuticular auricular-like structures, the reduced right caudal ala in the male and the tuberculate ornamentation on the end of the female tail.

From the available literature the writer knows of five species of filaria (S.L.) which have been recorded from Suidae: *Filaria acutiuscula* Molin, 1858, from the abdominal cavity and subcutaneous tissue of the Pecarry; *Filaria bauchei* Raill. and Henry, 1911, from the lungs of a domestic pig, Annam; *Setaria bernardi* Raill. and Henry, 1911, from the peritoneal cavity of the domestic pig, Annam; *Setaria congolensis* Raill. and Henry, 1911, from the peritoneal cavity of wild pigs (probably *Phacochoerus porcus* L.) Congo, and *Setaria Rodhaini* v. d. Berghe and Vuylsteke, 1936, from *P. porcus*, Belgian Congo. *F. acutiuscula*, according to van Thiel (1926), has a tubular and chitinised buccal capsule, but the female tail is not ornamented and the vulva is 1·5 mm. from the anterior extremity. *F. bauchei* attains a length of 22 cms. in the female, has no lips or buccal capsule, the vulva is situated about 1 mm. from the anterior extremity, and the tail appears to be devoid of ornamentations. The three species of Setaria are provided with typical peribuccal cuticular rings, and consequently cannot be confused with the species described above.*

From the information available, it would appear that the parsite described above does not affect the health of the host, and, except for the unsightly nature of the cysts, the writer does not think that humans would be exposing themselves to any danger should such infected meat be eaten by them.

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* According to Sandground (1933) *Setaria congolensis* and *S. bernardi* are cospecific, the former name having priority.

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Whipworms from South African Ruminants.

By R. J. ORTLEPP, Section of Parasitology, Onderstepoort.

UNTIL quite recently it had been taken for granted that the common whipworms of South African Ruminants belonged to the species *Trichuris ovis* (Abildgaard, 1795). Baylis (1932), however, found that *T. globulosa* (v. Linstow, 1906) was quite common among South African cattle, sheep and goats and thought that it would be found to be widely distributed in East and South Africa. This appears to be quite correct, for materials, from various localities in South Africa, in the collection of this institute, which had been identified as *T. ovis*, have on re-examination proved to belong practically all to the species *T. globulosa*; most striking, however, is that among this material there is not a single example of *T. ovis* as redescribed by Baylis (1932) and by Chandler (1930). This re-examination also showed that the whipworms from the Springbok and Blesbok belong to a hitherto undescribed species and that some specimens obtained from a goat and mixed with other goat material and used for class demonstrations, also belonged to a hitherto undescribed species. In the ensuing pages some remarks are passed on the morphology of *T. globulosa*, and the new species mentioned above are described in addition to a new species recently collected on a single occasion from an ox from the Barberton district of Transvaal.

TRICHURIS GLOBULOSA (v. Linstow, 1901).

This species appears to be the commonest species in South African Ruminants, and the materials in the collection of this institute were obtained from cattle, sheep, goats, sable antelope and a camel; an imported Nylghiae (*Boselaphus tragocamelus*), killed soon after its arrival from India, also harboured this parasite. All this material agrees in all essentials with the descriptions recently given by Baylis (1932), Gebauer (1932) and Sprehn (1927). The last two authors definitely state that a distinguishing feature of this parasite from *T. ovis* (Abild. 1795) is that the spicular sheath terminates in a rounded swelling in *T. globulosa* whereas in *T. ovis* it is melon-shaped. In the writer's material there are specimens showing no swelling, i.e. the sheath is only partially everted; a rounded swelling in which the tip of the sheath is not everted; and specimens with fully everted sheaths in which the swelling is rounded or melon-shaped and is terminated by a smooth "mouth piece" fitting closely to the spicule. The writer is quite satisfied that all these specimens are the same; the nature of the spicule is similar in all, being robust with large "flares" at their proximal ends and showing a slight thickening towards their distal ends and then thinning to end in a sharp point; the distal end of the spicule thus has a sabre-like appearance (Fig. 1); also the spines on the sheath are large towards its distal end and

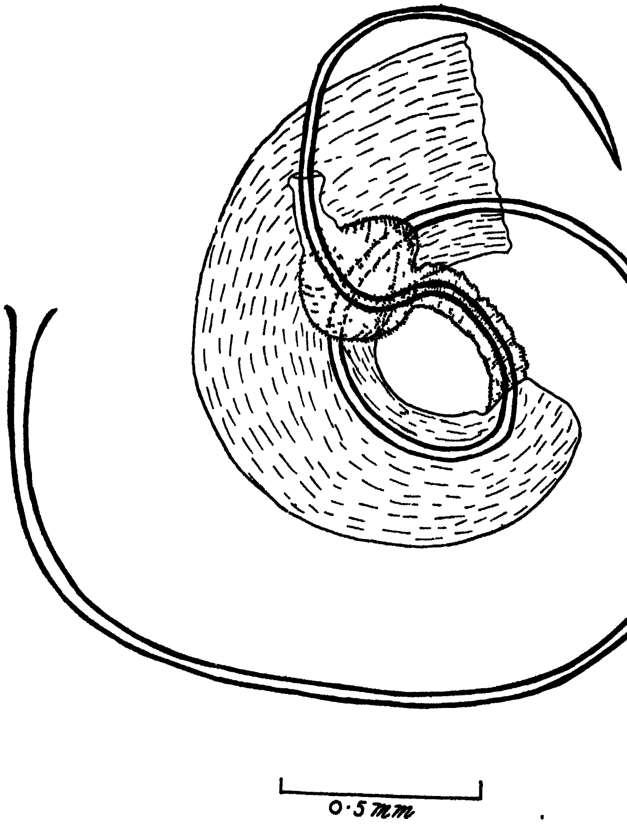

Fig. 1 *Trichuris globulosa*, posterior extremity of male.

become smaller towards its proximal end. . In addition all the females examined showed the characteristic structure of the vagina described and figured by Baylis. The variations in the lengths of the spicules observed by the writer agree with those of the above mentioned authors: They were from 3·8 to 5·7 mm. long by 0·035 to 0·043 mm. broad; the breadth also agrees with Baylis' findings but is slightly thicker than Gebauer's, and about half the thickness given by Sprehn (0·08-0·09 mm.).

The spines on the sheath were found to vary from 0·0175 to 0·019 mm. for the large spines on its distal end and from 0·006 to 0·007 mm. for the small spines at its proximal end.

With regard to the internal male genitalia, the cloaca was found to vary from 2·2 to 2·5 mm. in length and the spicular diverticulum joined it from 1·2 to 1·8 mm. from its external opening; the ejaculatory dust was from 5·9 to 7·8 mm. long and the vas deferens from 4·9 to 6·2 mm. long; a slight constriction joined these two parts. The testes, which terminated at about the level of the proximal end of the cloaca, was straight in the region of the ejaculatory duct, but was thrown into conspicuous dorso-ventral folds for its whole length opposite the vas deferens.

TRICHURIS BARBERTONENSIS sp n.

This species, of which 4 males and 5 female specimens were available, were collected from an ox from the Barberton district of the Transvaal. These were the only whipworms collected from this animal.

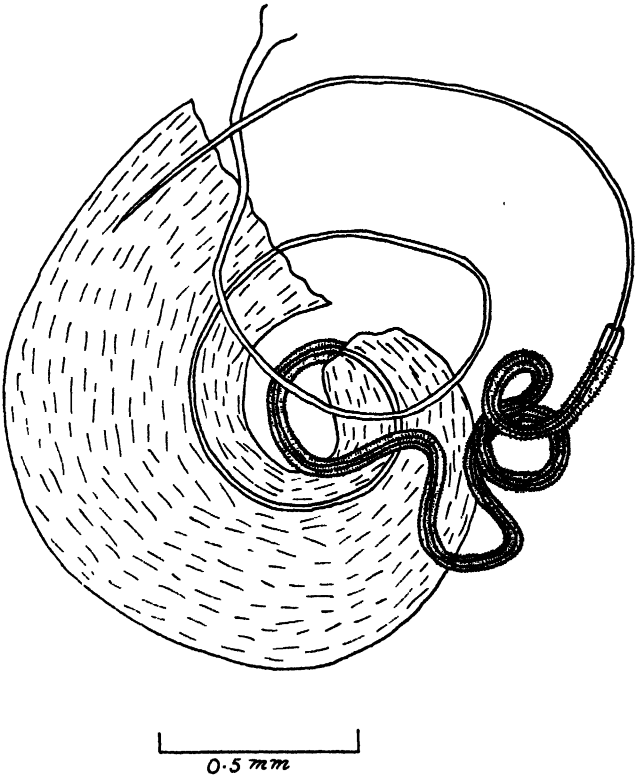

Fig. 2. *Trichuris barbertonensis* sp. n.; posterior extremity of male.

Superficially it is very similar to *T. globulosa*, the thick portion of the body being 11 to 13 mm. long in the male and 14 to 16 mm. long in the female by about 0·5 mm. thick in the latter.

The spicule is long and slender and in the four males measured 6·83, 6·92, 7·12 and 7·3 mm. with a maximum thickness of 0·014 to 0·016 mm. in its middle (Fig. 2);at their proximal ends, just behind the head, they were from 0·025 to 0·03 mm. thick; they decrease uniformly towards their distal ends to end in sharp tips. The sabre-like swelling seen in *T. globulosa*, and also described for *T. ovis* by Baylis, is absent. The sheath when fully extended has a uniform diameter and does not terminate in a bulb. It is very long and may attain a length of 2·7 mm. It is densely covered by minute spines which are, however, absent on its distal end or " mouth piece ", they are largest at its proximal end, reaching a length of 0·01 mm., and smallest at its distal end, where they are only 0·005 mm. long.

The cloaca is relatively long being up to 3·8 mm. long and it is joined by the spicular diverticulum at the junction of its 1st and 2nd proximal quarters. The ejaculatory duct is from 7·7 to 8·7 mm. long and a slight constriction joins it to the vas deferens which is 3·7 to 3·9 mm. long. The testes is convoluted opposite the vas deferens and straight opposite the ejaculatory duct and terminates at about the level of the proximal end of the cloaca.

The vagina is much convoluted and has a more or less even diameter throughout (Fig. 3); its distal end is everted through the vulva and is devoid of spines; instead the cells forming its lining are dome-shaped thus giving this portion a tuberculate appearance.

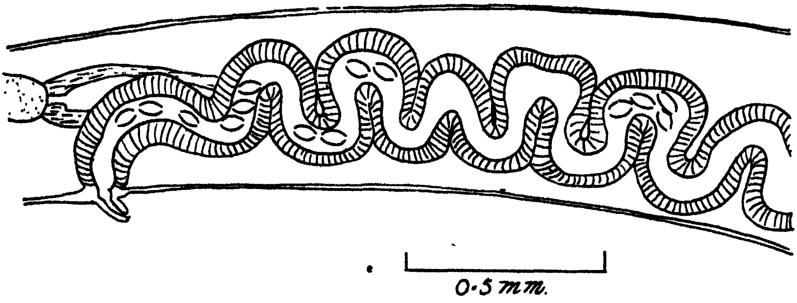

Fig. 3. *Trichuris barbertonensis* sp. n.; vagina.

The eggs are of the usual shape and vary in size from 0·046 to 0·052 mm. long by 0·022 to 0·024 mm. broad including the plugs.

Host: Ox.

Habitat: Caecum.

Locality: Barberton, Transvaal.

Types in the Helminthological Collection, Onderstepoort.

Specific diagnosis.

Trichuridae resembling *T. globulosa* but having a spicule up to 7·3 mm. long and evenly tapering towards its tip; spicule sheath long and slender and not terminating in a bulb. Cloaca up to 3·8 mm. long; vas deferens about half the length of the ejaculatory duct; and vagina simple, convoluted and not spined.

Caecum, Ox, Transvaal.

Discussion.

This species may be easily distinguished from both *T ovis* and *T globulosa* by the slender spicule devoid of a distal swelling; the long and slender spicular sheath not terminating in a bulb; the simple and convoluted vagina without internal spines; the very much longer cloaca and in that the vas deferens is only about half the length of the ejaculatory duct.

The material from a Uganda Bull, identified by Baylis (1932) as *T. ovis* and having a spicule 7·2 mm. long by 0·0175 mm. thick probably belongs to the above described species.

TRICHURIS ANTIDORCHI sp. n.

This species is represented in the collection by 10 males and 18 females from the caecum of the Springbok and one male and one female from the caecum of a Blesbok. Mönnig (1932) had identified the material from the Blesbok as *T. ovis* and that from the Springbok (1933) as *T. globulosa*. In size and general appearance they are indistinguishable from *T. globulosa*; differences are only evident when the internal organs are examined.

The spicule is remarkable for its robustness and by its weak cuticularization (Fig. 4). It varies in length from 5·43 to 6·5 mm. with an average middle thickness of 0·053 mm. It maintains a fairly even thickness throughout its length and just before its distal termination it suddenly narrows down to end in a fine point. When cleared in lacto-phenol or creosote the spicule can hardly be traced in the body and does not stand out as is the case in *T. globulosa*. The spicular sheath, when fully everted, terminates in a large globular swelling covered by numerous very minute spines from 0·003 to 0·004 mm. long; this swelling is drawn out into a smooth " mouth piece " which closely invests the spicule; the remaining portion of the sheath is tubular and is covered by backwardly directed spines which increase in size towards its proximal end; here the largest spines in the different males are from 0·01 to 0·014 mm. long. The cloaca, as in the preceding species, is also relatively long, being from 3·5 to 3·75 mm. long; the spicular diverticulum, however, joins it more posteriorly, 1·9 to 2·3 mm. from its external opening. The ejaculatory duct is relatively short being from 4·5 to 6 mm. long and the vas deferens, in proportion to it, is relatively long, 3·7 to 4·5 mm. long. A slight constriction joins these two parts. The testis is convoluted opposite the vas deferens and more or less straight opposite the ejaculatory duct, and it terminates just posterior of the anterior limit of the cloaca.

The vagina shows a very characteristic structure in that its distal half is telescoped into itself from two to five times, the usual number of telescopings being three (Fig. 5). This characteristic appears to be a constant feature as it was present in all the females; no such characteristic has so far been recorded in the literature dealing with whipworms. The internal surface of the vagina up to the vulva, and also that portion following the telescopings, is lined

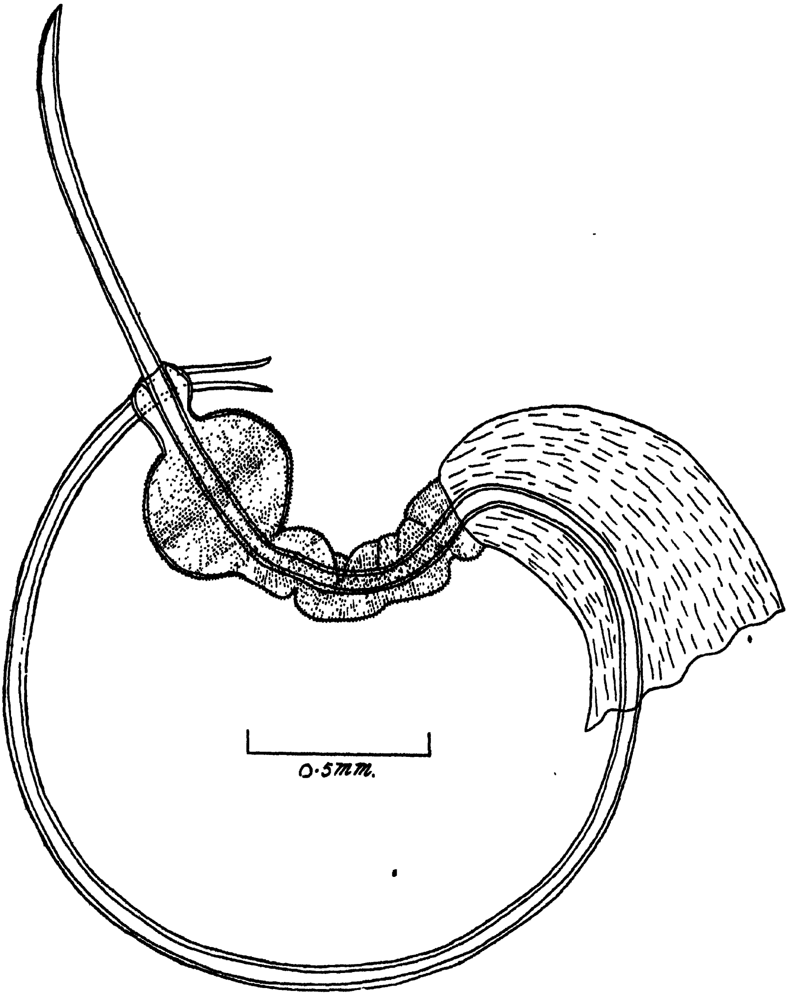

Fig. 4. *Trichuris antidorchi* sp. n.; posterior extremity of male.

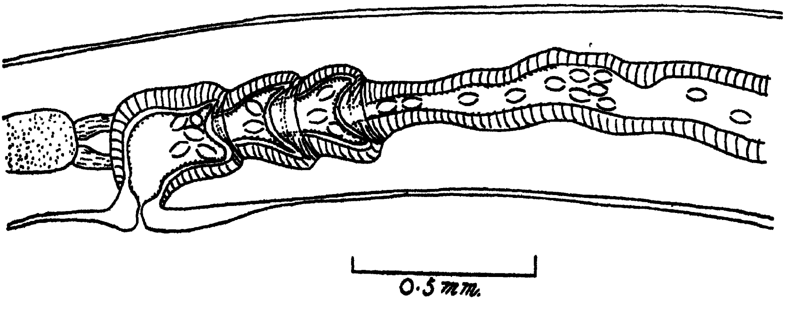

Fig. 5. *Trichuris antidorchi* sp. n.; Vagina.

by very minute spines; the rest of the vagina up to the uterus is lined by large and prominent columnar cells which project freely into its lumen and whose tips appear to be weakly cuticularized. In no case was the vagina everted through the vulva.

The eggs are oval and vary in size from 0·054 to 0·056 mm. long, including the plugs, by 0·027 to 0·029 mm. broad.

Host: *Antidorcas marsupialis* and *Damaliscus albifrons.*
Habitat: Caecum.
Locality: Theunissen, Orange Free State.

Types in Helminthological Collection, Onderstepoort.

Specific diagnosis.

Trichuridae resembling *T. globulosa* but having a robust, weakly cuticularized spicule up to 6·5 mm. long, and not sabre shaped posteriorly; spines on spicular sheath decrease in size posteriorly; distal portion of vagina telescoped. Cloaca relatively long and vas deferens about ¾ length of ejaculatory duct.

Caecum, Springbok and Blesbok, O.F.S.

Discussion.

The nature of the spines on the spicular sheath appear to be similar to those found in *T. ovis*, but apart from this similarity the writer's species may be easily distinguished from other ruminant whipworm by the robust and feebly cuticularized spicule without a distal enlargement and by the telescopings in the distal portion of the vagina.

TRICHURIS PARVISPICULUM sp. n.

In a bottle containing specimens of *T. globulosa* from a South African goat, there was present a fair number of individuals of this species. Macroscopically there were no distinguishing features whereby it could be separated from *T. globulosa.*

The thick portion of the body is 12 to 14 mm. long in the male by about 0·4 mm. thick; in the female the length is 12·5 to 14 mm. by 0·4 to 0·5 mm. thick.

In the male the most striking characteristic is the remarkably short spicule which terminates in a bluntly rounded tip (Fig. 6); in ten males the following spicular lengths were obtained:—0·85 mm., 0·87 mm., 0·88 mm., 0·89 mm., 0·93 mm., 0·95 mm., 1·06 mm., 1·07 mm. and 1·07 mm. with an initial thickness just behind the "flare " of 0·014 to 0·02 mm., and 0·006 to 0·008 mm. at the tip. The fully everted spicular sheath is relatively short being only about 0·2 mm. long; its distal end becomes enlarged to form a melon-shaped swelling provided with a smooth " mouth-piece "; its proximal end has a diameter of about 0·032 mm. and across the swelling may reach 0·056 mm.; the entire sheath, except for the " mouth-piece " is studded by very minute spines which are slightly larger on the swelling (0·002 mm.) than those at its base (0·0015 mm.).

On either side of the cloaca there is a prominent conical papilla similar to that found in *T. gazellae* Gebauer, 1933. The cloaca is short being from 1 to 1·01 mm. long and it is joined by the spicular diverticulum 0·48 to 0·56 mm. from its external opening. The ejaculatory duct is relatively very long being from 8 to 8·2 mm. long and the vas deferens is 4·8 to 5 mm. long; these two parts are joined to each other by a narrow constriction, 0·01 mm. long. Opposite the vas deferens the testes is thrown into closely packed dorso-ventral loops; it terminates just posterior of the anterior limit of the cloaca.

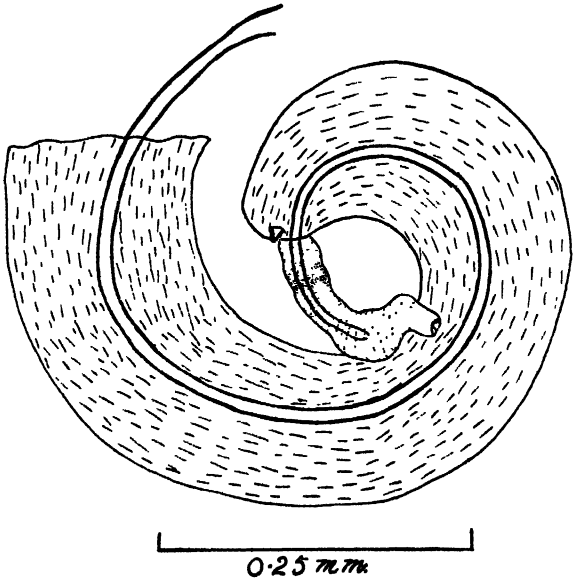

Fig. 6. *Trichuris parvispiculum* sp. n: ; posterior extremity of male.

In the females the distal end of the vagina is in nearly all cases slightly everted through the vulva, and its lumen is lined by very minute spines. Its distal half may pass straight down the body, but a few slight curves are generally present (Fig. 7); the middle portion of the vagina is slightly thickened giving it a slight club-shaped appearance; behind this thickening it is more wavy until it joins the uterus.

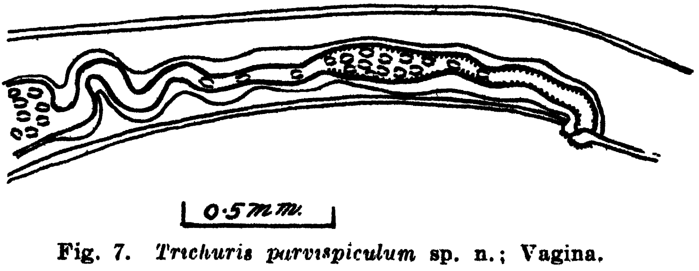

Fig. 7. *Trichuris parvispiculum* sp. n.; Vagina.

The eggs, including the plugs, are 0·044 to 0·046 mm. long by 0·023 to 0·025 mm. broad.

Host: *Capra hircus.*
Habitat: Caecum.
Locality: South Africa.

Types in the Helminthological Collection at Onderstepoort.

Specific diagnosis.

Trichuridae resembling *T. globulosa* externally but having a small spicule up to 1·07 mm. long, but generally less than 1 mm. long, ending in a bluntly rounded tip; a pair of caudal papillae at sides of cloacal aperture; Cloaca about 1 mm. long; ejaculatory duct 8 mm. long and over; vagina simple, more or less straight and provided with minute spines.

Caecum, Goat, South Africa.

Discussion.

Of the whipworms from ruminants this species, because of its short spicule, appears to be closely related to *T. discolor* (v. Linstow, 1906), and *T. spiricollis* Solomon, 1932 and to a lesser extent to *T. gazellae* Gebauer, 1933. It agrees with v. Linstow's species in that the tip of the spicule is bluntly rounded, but the spicule is nearly twice as long as in the writer's species and there are no caudal papillae. Gebauer's species has a spicule up to 4·15 mm. long but it also ends bluntly, and in addition two prominent caudal papillae are present. Solomon's species has a spicule the same length as the writer's species, but differs in that the tip is spatulate; also the body carries anterior cuticular " plagues ", the cloaca is only from 0·2 to 0·3 mm. long as against about 1 mm. in the writer's species, and caudal papillae are absent.

SUMMARY.

The occurrence and morphology of *Trichuris globulosa* are discussed and three new species are described, namely *T. barbertonensis* from cattle, *T. antidorchi* from springbok and blesbok and *T. parvispiculum* from goats.

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Onderstepoort Journal of Veterinary Science and Animal Industry, Volume 9, Number 1, July, 1937.

Printed in the Union of South Africa by the
Government Printer, Pretoria.

The Approximate Distribution of the Genus Glossina.

By H. H. CURSON, Deputy Director of Native Animal Husbandry, Pretoria, and W. O. NEITZ, Section of Protozoology and Virus Diseases, Onderstepoort.

THE compilation of the map accompanying this paper arose from a desire to demonstrate at a glance to veterinary students the approximate distribution of the genus *Glossina*, a knowledge of which is especially useful in discussing problems such as the distribution of cattle. If we consult the literature of the past fourteen years nowhere do we find a map giving the information in this form. Steffan (1922) is more concerned with the approximate limits of each of the four groups, viz. *fusca*, *brevipalpis*, *morsitans*, and *palpalis*, and apart from the intersections of the many lines indicating limits of distribution, a clear idea of the Glossina and non-Glossina regions is not possible. Austen and Hegh (1922) while furnishing a written description of the general geographical distribution do not supply a map. Newstead (1924) gives details concerning the distribution of the several species and to avoid the maze and intersecting lines, four maps are employed. Finally Hegh (1929) in dealing with the same aspect includes a map, which indicates the northern and southern boundaries, but within this area there are thousands of square miles of Glossina-free territory eminently suitable for cattle.

Admittedly the task is difficult especially on a small scale map, but it is this feature we wish to represent in this paper.

Accordingly an appeal for data was issued to the administrations of the various territories concerned, and thanks to the generous response it has been possible to compile the accompanying map. In the following section the replies are commented upon where necessary.

Country and (Reference).	*Comments.*
French West Africa and French Equatorial Africa. (*Letter* 1250 *of* 14.5.35 *from the Director of the Institut National d'Agronomie Coloniale—Ministry of Colonies—to the Director of Veterinary Services, Pretoria.*	Accompanying the letter was a map showing the approximate limit of distribution of the genus Glossina. This was prepared by the Entomological section of the Ministry of Colonies in collaboration with Professor Roubaud of the Institut Pasteur.

THE APPROXIMATE DISTRIBUTION OF THE GENUS GLOSSINA.

Country and (Reference).	*Comments.*
Sierra Leone. (*Letter A/76/29 of* 28.5.36 *from Colonial Secretary to Director Veterinary Services, Pretoria*).	Reference was made to Dr. Simpson's researches published in *The Bulletin of Entomological Research* IV, 1913, from which it would appear that the genus Glossina is widely distributed.
Gold Coast. (*Letter* 573/30/1911 *of* 24.3.36 *from Principal Veterinary Officer to Director of Veterinary Services, Pretoria.*	While the accompanying map indicates widespread distribution, this is confirmed by the following in the letter: "With regards Glossina you may take it that the whole country is infested". Reference is made to *G. palpalis*, *G. tachinoides*, *G. morsitans*, *G. submorsitans*, and *G. longipalpis* being prevalent.
Nigeria. (*Letter V.* 89/1932/108 *of* 1.1.36 *from Chief Veterinary Officer to Director of Veterinary Services, Pretoria.*)	The map received from Nigeria shows an extensive distribution. In fact the letter states that in "Northern Nigeria the infection is widespread, especially during the rains, but is light. We estimate that 33 per cent. of the cattle in the North are infected, but it is only in *G. morsitans* country where the mortality is heavy."
Gambia. (*Letter* 607/1928 *of* 16.6.36 *from Colonial Secretary to Director of Veterinary Services, Pretoria.*)	"Tsetse flies are found along the whole length of the Gambia river, except in Bathurst where there is no suitable vegetation and along the coast of the estuary. They are also to be found in all patches of thick bush where there is a branch river . . . or swamps lasting through the dry season". *G. palpalis* is the commonest type of tsetse, but *G. tachinoides* and *G. morsitans* are also reported. (Letter 607/1928 of 16.6.36). See map accompanying Dr. J. J. Simpson's report on "Entomological Research in British West Africa—Gambia". *Bull. Entom. Res.* Vol. II, Part 3, 1912.
Portuguese Guinea and Angola. (*Letter* 82 *of* 6.4.35 *from the Chief of the Reparticao de Saude, Ministry of Colonies, to Director of Veterinary Services, Pretoria.*)	While the names of *G. palpalis*, *G. longipalpis*, *G. submorsitans*, and *G. fusca* are merely mentioned for Portuguese Guinea, the approximate distribution of *G. palpalis* and *G. morsitans* in Angola is traced on a map.
Sudan. (*Letter* 13–*A*–6 *of* 3.11.34 *from Director of Veterinary Services, Khartoum, to Director of Veterinary Services, Pretoria.*)	A copy of the paper "Distribution of Tsetse flies in the Sudan" by Bedford, H.W., in the *Bull. Entom. Res.* XXI (3) Oct. 1930 was supplied.
Uganda.	The approximate areas occupied by the genus Glossina were indicated on a map.
Kenya. (*Letter Fly/1/ of* 16.10.34 *from Chief Veterinary Officer to Director of Veterinary Services, Pretoria.*)	A map "prepared a few years ago by, I believe, the Medical Entomologist . . . Nairobi" was received. It indicated record of *G. palpalis*, *G. pallidipes*, *G. brevipalpis* but added to this by the Veterinary Entomologist, Kabete, were further details regarding "*G. swynnertoni*, *G. fuscipleuris*, *G. austeni* and *G. longipennis*." A more suitable map prepared by Dr. Lewis, Veterinary Entomologist, Kabete, was supplied later. This included "the small border areas" of Abyssinia and Italian Somaliland.

Country and (*Reference*).	*Comments.*
Tanganyika. (*Letter SE* 38/360.0 *of* 12.11.34 *from Senior Entomologist to Director of Veterinary Services, Pretoria.*)	(1) " I (i.e. Potts, W. H.) am hoping shortly to publish a map showing the details of the distribution of the different species of Glossina ". In the meantime, thanks to Dr. John Phillips, now of the University of the Witwatersrand, Johannesburg, we received a photograph of a map compiled from data collected by Messrs. C. F. M. Swynnerton and W. H. Potts between 1922-1928. This refers in particular to the following species *G. morsitans, G. pallidipes, G. brevipalpis, G. austeni* and *G. swynnertoni*. (2) Potts recommended the perusal of the Glossina map accompanying the *Annual Report of the Veterinary Department*, 1925.
Belgian Congo. (*Letter* 5/1210 *of* 3.10.35 *from the Ministry of Colonies, Brussels, to Director of Veterinary Services, Pretoria.*)	Accompanying the reply was a copy of the *Bull. Agr. du Congo Belge XXV* (4) Dec. 1934, which contains the interesting paper by E. Hegh " Les quatorze espécés de tsétsés du Congo Belge ". Thanks to Dr. M. Moreau (letter 253 of 7.6.36) of Elizabethville a map showing the distribution in Katanga of *G. palpalis, G. morsitans, G. pallidipes, G. brevipalpis* and *G. fusca* was also received. Further information regarding this territory has been obtained from " *Manson's Tropical Diseases* " which only indicates the areas in which human sleeping sickness occurs.
Nyasaland. (*Letter* 681/63/34 *of* 30.10.34 *from Acting Chief Veterinary Officer to Director of Veterinary Services, Pretoria.*)	(1) " A map . . . showing the distribution of tsetse-fly " was received. This " has been made from records kept " by the Veterinary Department at Zomba. (2) " Practically the only species found is *G. morsitans*."
Northern Rhodesia. Southern Rhodesia. Bechuanaland Protectorate.	See Maps C, B and A in " Distribution of Glossina in the Bechuanaland Protectorate " in the 18*th Report of the Director of Veterinary Services and Animal Industry*, 1932.
Mozambique. (*Letter* 191 *of* 16.5.35 *from Acting Director of Veterinary Services, Lourenco Marques to Director of Veterinary Services, Pretoria.*)	A detailed map showing the distribution of the genus Glossina was received. Also a copy of the publication (1930) *Distribuicao geografica das glossina em Mocambique* describing Dr. S. Napoles' investigations in the District of Mozambique.
Zululand.	See Map 1 in " Nagana in Zululand " published in the 13*th and* 14*th Reports of the Director of Veterinary Education and Research*, 1928.
Spanish Guinea and Liberia. No information could be obtained.	The territory is left unshaded.

CONCLUSION.

It is granted that even within the area showing the distribution of the genus Glossina there are regions, e.g. in the Gold Coast and Cameroons, where tsetse flies are absent and cattle abound (see " A Contribution to the Study of African Cattle ", by Curson, H. H. and Thornton, R. W.). Nevertheless we submit that the general geographical distribution as here shown is at least as satisfactory as that given in the past.

ACKNOWLEDGMENT.

We desire to express our indebtedness to all those who assisted in this compilation. Obviously without their co-operation this task would have been impossible. A special word of appreciation is due to Messrs. C. G. Walker and T. Meyer for the drawing and photographic work respectively.

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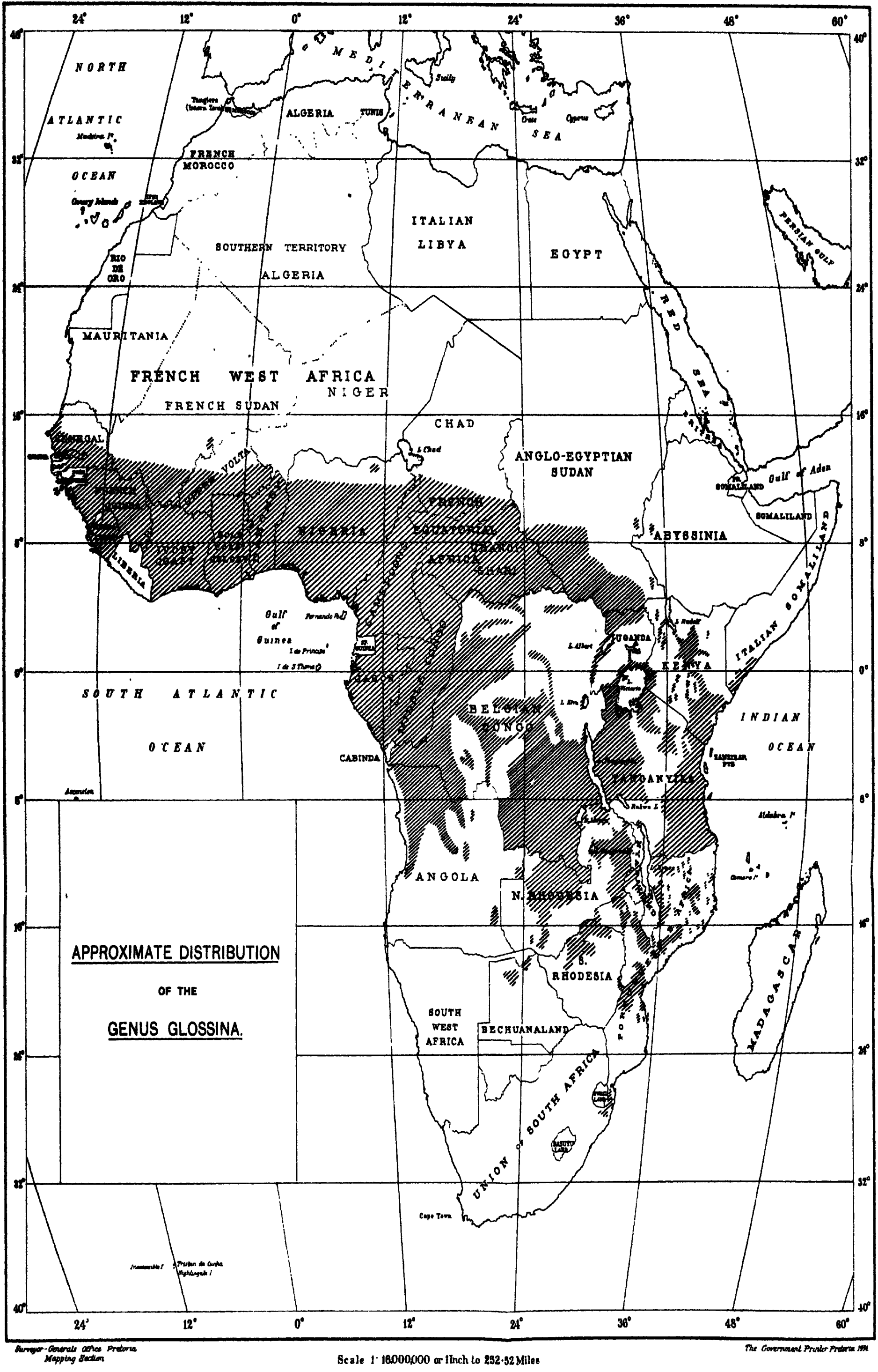
AFRICA

DEPARTMENT OF AGRICULTURE & FORESTRY



APPROXIMATE DISTRIBUTION

OF THE

GENUS GLOSSINA.

Surveyor-Generals Office Pretoria,
Mapping Section

The Government Printer Pretoria 1931

Scale 1: 16,000,000 or 1 inch to 252·52 Miles

100 50 0 500 1000 1500 2000
Miles

Section V.

Poisonous Plants.

STEYN, DOUW G. ... ... Experiments with plants alleged to be used as abortifacients and ecbolics by natives 107

STEYN, DOUW G. ... ... Recent investigations into the toxicity of known and unknown poisonous plants in the Union of South Africa, VII. 111

Onderstepoort Journal of Veterinary Science and Animal Industry, Volume 9, Number 1, July, 1937.

Printed in the Union of South Africa by the
Government Printer, Pretoria.

Experiments with Plants alleged to be used as Abortifacients and Ecbolics by Natives.

By DOUW G. STEYN, Section of Pharmacology and Toxicology, Onderstepoort.

THE undermentioned plants, which are alleged to be used as abortifacients and ecbolics by the natives of Uganda, were submitted by the Director of Medical Services, Uganda, for investigation. It is stated that the pregnant women drink infusions prepared from about a handful of the leaves and roots of the plants and that their use is often attended with disastrous results.

In the experiments conducted at Onderstepoort the infusions were prepared by extracting 40 gm. of ground dry plants with 300 c.c. of tap-water for two hours at 50° C. On each day of the experiment the infusions were freshly prepared before dosing. As the infusions apparently had no effect on the pregnant rabbits the plant material itself was administered also with negative results.

CUCURBITACEAE.

Momordica foetida Schum.

Registered No.: O.P. 6856; 30.1.1936.

Common names: Zulu—inTshungu.
Uganda—Luyula.

State and Stage of Development.—Dry and in the flowering stage.

In South Africa the Zulus take an infusion or a decoction of the vines of the plant as a gastro-intestinal sedative (Bryant, 1909).

Pregnant Rabbit A (2·32 *Kg.*).—Received infusion equivalent to 20 gm. of dry plant daily for six days and 10 gm. of dry plant as such daily for three days.

Result.—Normal fully developed young were born on the tenth day of the experiment.

Pregnant Rabbit B (2·5 *Kg.*).—Received the same quantity of infusion and the same amount of dry plant material as rabbit A.

Result.—Normal fully developed young were born on the eleventh day of the experiment.

I might mention that on a previous occasion a non-pregnant rabbit received 100 gm. of the fresh immature fruit per stomach-tube within six hours without suffering any ill effects.

LABIATAE.

Leonotis americana.

Registered No.: O.P. Herb No. 6856 A; 30.1.1936.

Common name: Uganda—Kifumafuna.

State and Stage of Development.—Dry and in flowering stage.

Pregnant Rabbit A (2·3 *Kg.*).—Received infusion equivalent to 20 gm. of dry plant daily for four days.

Result.—Animal gave birth to three normal fully developed foetuses four hours after the fourth dose had been administered.

Pregnant Rabbit B (2·07 *Kg.*).—Received infusion equivalent to 20 gm. of dry plant daily for six days and 10 gm. of dry plant as such daily for three days.

Result.—Normal fully developed young were born on the sixteenth day of the experiment.

Pregnant Rabbit C (2·3 *Kg.*).—Received the same quantity of infusion and dry plant as such as Rabbit B.

Result.—Normal fully developed young were born on the fifteenth day of the experiment.

MALVACEAE.

Abutilon indicum Don.

Registered No.: O.P. Herb No. 6856 B; 30.1.1936.

Common name: Uganda—Kifura.

State and Stage of Development.—Dry and in flowering stage.

Pregnant Rabbit A (2·65 *Kg.*).—Received infusion equivalent to 20 gm. of dry plant daily for six days and 10 gm. of dry plant as such daily for three days.

Result.—Normal fully developed young were born on the thirteenth day of the experiment.

Pregnant Rabbit B (2·5 *Kg.*).—Received the same quantity of infusion and dry plant as Rabbit A.

Result.—The animal gave birth to normal fully developed young on the fifteenth day of the experiment.

The plant is indigenous to southern and eastern Asia, where its seed and bark are used medicinally. Its fibre is used commercially (Wehmer, 1931).

DISCUSSION.

From the above experiments it would seem that *Momordica foetida*, *Leonotis americana*, and *Abtilon indicum* in the dry state and flowering stage do not act as abortifacients on pregnant rabbits. The results of the above experiments, however, do not warrant the conclusion that pregnant women will not be affected by these plants as the pregnant human uterus may react to these plants in a way different from that of the pregnant rabbit uterus.

It also appears that the plants have no toxic effects on rabbits as they were administered in fairly large quantities.

Dr. C. Rimington, Onderstepoort, conducted preliminary tests for alkaloids and found slight positive reactions with *Leonotis americana* and *Momordica foetida*. No alkaloids were detectable in *Abutilon indicum*.

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Recent Investigations into the Toxicity of known and unknown Poisonous Plants in the Union of South Africa VII.

By DOUW G. STEYN, Section of Pharmacology and Toxicology, Onderstepoort.

(Continued from *Onderstepoort Journ. Vet. Sc. and Anim. Ind.* Vol. 6, No. 2, 1936.)

AIZOACEAE.

GALENIA AFRICANA L.

Registered number: O.P.H. No. 15851; 27.11.34. N.H. No. 19566.

Common name: Kraalbos.

Origin: Willowmore, C.P.

State and stage of development: Dry and in flowering stage.

Goat 41205 (1 year old, 18 Kg.): Received 50 gm. of dry plant per stomach-tube daily* from 8.4.35 to 30.6.35. The animal received a total amount of 3·6 Kg. of dry plant (=9 Kg. of fresh plant.) At no time were any symptoms of poisoning discernible. The animal was killed on 16.7.35 and the organs examined. No macroscopic or microscopic lesions were detectable in the internal organs.

Goat 41204 (1 year old, 15 Kg.): Received the same amount of dry plant as Goat 41205, also with negative results. No macroscopic or microscopic lesions were detectable in the internal organs of this animal, which was killed on 3.9.35.

Goat 41665 (1 year old, 20 Kg.): Received the same quantity of dry plant as Goat 41205 without developing any symptoms of poisoning.

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* Except Sundays.

Goat 42918 (1 year old, 16 Kg.): Received 100 gm. of dry plant per stomach-tube daily from 8.4.35 to 30.6.35. At no time were any symptoms of poisoning discernible. The animal was killed on 15.7.35 and no macroscopic or microscopic lesions were detectable in any of the internal organs.

The animal received a total amount of 7·2 Kg. dry plant equivalent to 18 Kg. of fresh plant.

Goat 39821 (1 year old, 18 Kg.): Received the same quantity of dry plant as Goat 42918 with the same result. The animal was killed on 3.9.35 and no macroscopic or microscopic lesions were detectable in the internal organs.

Goat 38665 (1 year old, 15 Kg.): Received the same quantity of dry plant as Goat 42918 without developing any symptoms of poisoning.

Goat 41149 (3 years old, 26 Kg.): Received 300 gm. of dry plant per stomach-tube daily from 8.4.35 to 30.6.35. No symptoms of poisoning were discernible. The animal was killed on the 16.7.35 and no macroscopic or microscopic lesions were detectable in the internal organs. The total amount of dry plant administered to the animal is 21·6 Kg. equivalent to 54 Kg. of fresh plant.

Goat 42917 (2 years old, 30 Kg.): Received the same quantity of dry plant as Goat 41149 without developing any symptoms of poisoning.

Goat 42916 (3 years old, 33 Kg.): do.

All the above animals increased in weight in the course of the experiment. Those goats that were not killed were kept under observation for about six months after discontinuation of the drenching.

Discussion.

Many farmers in the Southern and Western Karroo maintain that *Galenia africana* is the cause of "waterpens" (hydroperitoneum) in goats. All we can conclude from this experiment is that the dry plant administered in the quantities mentioned above during a period of twelve weeks apparently has no ill-effects on goats. We have to consider the possibility of (*a*) the dry plant being less toxic than the fresh plant, (*b*) the plant having toxic effects when eaten over prolonged periods in amounts greater than those administered to the above experimental animals, and (*c*) the plant varying in toxicity in its different stages of development and in different years.

The only reliable way to determine whether the "kraalbos" is the cause of "waterpens", or not, is to conduct grazing experiments in the areas where the plant abounds. The experimental animals could then be forced to ingest much larger quantities of the plant than could be administered to them by stomach-tube.

ASCLEPIADACEAE.

PERGULARIA GARIEPENSIS N.E. BR.

Registered number: O.P.H. No. 9282, 11.12.35.

Common name: —

Origin: R. Schwarzkopf, Breckhorn West, Marienthal, S.W.A.

State and stage of development: Wilted and in early fruiting stage.

Sheep 40886 (4 years old, 50 Kg.): Received 250 gm. of the wilted plant per stomach-tube at 11 a.m. on 11.12.35. At 5 p.m. the animal was examined and no symptoms of poisoning were discernible. It was found dead in the stable at 7 a.m. the following morning.

Post-mortem appearances.—Interim—2 hours. Pronounced general cyanosis; pronounced injection of the subcutaneous blood vessels on the neck and front quarters; pronounced hyperaemia and slight oedema of the lungs; hyperaemia and degenerative changes in the liver; rumen distended with gas; and numerous pin-point haemorrhages in duodenal mucosa.

Histological examination of organs.—Fatty changes in the liver.

Sheep 39755 (4 years old, 45 Kg.): Received 250 gm. of the wilted plant per stomach-tube at 8.30 a.m. on 12.12.35. At 6 p.m. animal was restless walking about with short steps. It appeared stiff and paretic. There were pronounced laboured respiration and hoven. The pulse was accelerated but strong. The animal was found dead at 7 a.m. the following morning.

Post-mortem appearances.—In addition to the lesions described in sheep 40886 there was hyperaemia of the duodenal mucosa.

Sheep 40669 (Full mouth, 45 Kg.): Received 250 gm. of the dry plant per stomach-tube at 9 a.m. on 28.1.36.

No symptoms of poisoning were discernible until 6 p.m. and the animal was found dead at 7 a.m. the following morning.

Post-mortem appearances.—Pronounced general cyanosis; rumen markedly distended with gas; slight hydropericardium and hydroperitoneum; hyperaemia of the lungs with petechiae in the cervical portion of the trachea and extensive localised haemorrhage into the submucosa.

Rabbit (2·4 Kg.): Received 15 gm. of the dry plant per stomach-tube at 10 a.m. on 15.1.36.

16.1.36—apparently healthy. Another 30 gm. of dry plant in 2 doses. Within one hour after the second dose the animal developed weakness in the neck, an accelerated and weak heart-beat and progressive paralysis until it was apparently completely paralysed. Death occurred about three hours after the second dose.

Post-mortem appearances.—Hyperaemia of the lungs and liver.

SARCOSTEMMA VIMINALE R. BR.

Registered No.: O.P.H. No. 3298; 2.7.35.

Common names: Melktou, spantou—melkbos.

Origin: Komaggas Reserve, Namaqualand.

State and stage of development: Fresh plant with no flowers or fruits.

Sheep 42538 (25 Kg.): Received 350 gm. of the fresh vines per stomach-tube at 4 p.m. on 2.7.35.

The animal was found dead at 8 a.m. the following morning.

Post-mortem appearances.—Pronounced general cyanosis; rumen markedly distended with gas; pronounced injection of the subcutaneous vessels in the front quarters; subepicardial and intramyocardial haemorrhages; hyperaemia of and degenerative changes in the liver; pronounced hyperaemia and oedema of the lungs with extensive haemorrhage into the submucosa and mucosa of the trachea and bronchi.

Sheep 42488 (45 Kg.): Received 450 gm. of the fresh vines per stomach-tube at 9 a.m. on 4.7.35.

5.7.35—8 a.m.: Apathetic, swaying gait, accelerated and weak pulse, laboured respiration. At 11 a.m. the animal was prostrate with the head thrown backwards and the legs extended as in strychnine poisoning; twitching of the eye-balls; general cyanosis; accelerated and superficial respiration; groaning; champing; dilatation of the pupils; continual clonic contractions of neck muscles (head shivering); and hoven. The animal was killed in a state of unconsciousness at 4 p.m. on 5.7.35.

Post-mortem appearances.—General cyanosis; rumen distended with gas; hyperaemia of the lungs; hyperaemia of and degenerative changes in the liver; hyperaemia of mucosa at bifurcation of the trachea.

Rabbit A (2·5 Kg.): Received the following quantities of the fresh vines per stomach-tube without suffering any ill-effects: 4.7.35—30 gm.; 5.7.35—60 gm.; 6.7.35—30 gm.; and 8.7.35—50 gm.

Rabbit B (2·7 Kg.): Received 60 gm. of the fresh vines daily for four days without developing symptoms of poisoning.

COMPOSITAE.

DIMORPHOTHECA NUDICAULIS DC.

Registered No.: O.P.H. No. 6654; 27.9.35.

Common names: Jakkalsgras, ox-eye daisy, wilde wit magriet.

Origin: Sandberg, Clanwilliam district.

State and stage of development: Wilted plant in the flowering and seeding stage.

Hydrocyanic acid test (Guignard test).

(*a*) 10 gm. wilted leaves—strongly positive within 2 minutes.

(*b*) 10 gm. wilted leaves + chloroform—strongly positive within a few seconds.

(*c*) 10 gm. wilted leaves + emulsin—strongly positive within 1 minute.

In view of the large amount of hydrocyanic acid present in the plant it should be considered dangerous to stock.

HERTIA PALLENS (DC) O. KUNTZE.

(= OTHONNA PALLENS DC.)

Registered No.: O.P.H. No. 2747; 21.7.36.

Common names: Springbokbossie, vaalbos, dikkopbos.

Origin: Bestersput, Petrusburg district, O.F.S.

State and stage of development: In the post-seeding stage. The plant was dried in the shade.

Sheep 28519 (40 Kg.): Received 150 gm. of dry plant per stomach-tube on each of 21.7.36 and 22.7.36. 23.7.36 8 a.m.. Animal looked very ill; apathetic; fairly pronounced hoven; accelerated and strong pulse; temperature 99·4° F.; Bloody mucus exuding from both nostrils.

24.7.36: pronounced hoven; irregular and accelerated respiration; groaning; weak and accelerated pulse.

25.7.36: hoven; animal seemed "stupid" and took no notice of persons approaching her; resting nose on the ground when standing; swaying from side to side; accelerated and double breathing; death occurred at 10.45 a.m. on the 25.7.36.

Post-mortem appearances.—Pronounced general cyanosis; slight hydroperitoneum and hydropericardium; pronounced hyperaemia and slight oedema of the lungs; bloodstained mucus in the trachea and bronchi; subpleural emphysema in lungs; subendocardial haemorrhages in left ventricle; swelling and very pronounced fatty degeneration of the liver; cystic degeneration of the kidneys; pronounced dilatation of the heart; yellowish gelatinous infiltration of the subcutaneous tissues on the ventral aspect of the neck and the mesentery; slight acute catarrhal enteritis; stasis in caecum and colon with hyperaemia of the mucosa; the uterus contained foetus approximately three months old.

Histology.—Pronounced fatty degeneration of the liver with slight interstitial hepatitis. No specific changes in the kidneys, lungs, lymphatic glands and spleen. Slight fatty changes in the myocard.

Sheep 42972 (40 Kg.): Received 300 gm. of dry plant per stomach-tube at 9 a.m. on 21.7.36. At 4 p.m. the animal exhibited laboured respiration and was found dead at 7 a.m. the following morning.

Post-mortem appearances.—White froth exuding from both nostrils; rumen distended with gas; pronounced general cyanosis; injection of the subcutaneous blood vessels; pronounced oedema and slight hyperaemia of the lungs; hyperaemia of and haemorrhages in the bronchial and mediastinal lymph-glands; haemorrhages in the tracheal mucosa; coagulated blood could be squeezed from some bronchi.

Histology.—Liver—central necrosis and slight interstitial hepatitis. No specific changes in the kidney and myocard.

Discussion.

The symptoms and post-mortem appearances (especially the macroscopic appearance of the liver) resemble those seen in cases of "domsiekte" in sheep to a certain extent. It would not seem unreasonable to suggest that at least some cases of "domsiekte" may be due to poisoning with the plant, *Hertia pallens.*

SENECIO GLUTINOSUS THUNB.

Registered No.: O.P.H. No. 5785; 13.9.35.

Common name: Ragwort.

Origin: Hopetown, C.P.

State and stage of development: Fresh and in the flowering stage.

Sheep 43795 (four-tooth, 35 Kg.): Received 600 gm. of the fresh plant per stomach-tube daily from 13.9.35 to 17.9.35, followed by doses of 400 gm. of the wilted plant on each of 18.9.35 and 19.9.35 and by doses of 300 gm. of dry plant daily from 20.9.35 to 23.9.35.

No symptoms of poisoning were discernible except that the temperature rose to 104-106° F. on a few occasions.

Discussion.

The toxicity of this plant was investigated as donkeys, which were grazing on a camp on the banks of the Orange River near Orange River Station, where the author found the above species of Senecio growing plentifully, developed typical symptoms of "dunsiekte" (seneciosis).

The fact that the above experiment yielded negative results is for various reasons by no means proof that this plant is non-toxic. According to observations made in the field it appears definite that this *species of Senecio* may in certain circumstances produce seneciosis.

SENECIO LAEVIGATUS.

Registered No.: O.P.H. No. 3627; 12.7.35.

Common names: Ragwort, sheep thrive (Queenstown).

Origin: Middeldrift, C.P.

State and stage of development: Fresh and in flowering stage.

Sheep 43448 (4-tooth, 35 Kg.): Received a total of 700 gm. of the fresh plant on two consecutive days. Result: negative.

SENECIO SP.

(Sent to Botanic Gardens, Kew, for identification.)

Registered No.: O.P.H. No. 2016; 5.6.36.

Common name: —

Origin: Roodekuil Estates, P.O. Warmbaths, Transvaal.

State and stage of development: Fresh and in flowering stage.

Sheep 38890 (45 Kg. full mouth): Received 800 gm. of fresh plant daily (except Sundays) from 5.6.36 to 13.6.36 and 300 gm. of dry plant from 14.6.36 to 16.7.36, that is a total of 6·4 Kg of fresh plant and 8·4 Kg. of dry plant in the course of 42 days.

Sheep 40752 (51 Kg., full-mouth): Drenched as sheep 38890. Result: At no time did the animals exhibit any symptoms of poisoning. They were killed on 23.7.36 and no macroscopic or microscopic lesions were discernible in the internal organs.

Alkaloids isolated from species of Senecio—Isatidine and Retrorsine.

A small quantity of *isatidine* and *retrorsine* isolated by Mr. J. J. Blackie, Holyrood Road, Edinburgh, Scotland, from *Senecio isatideus* and *Senecio retrorsus* respectively was kindly sent to us by Professor G. Barger, Department of Medical Chemistry, University of Edinburgh, Edinburgh, for experimental purposes.

It was our intention to produce, if possible, with these alkaloids cases of chronic seneciosis (*dunsiekte*) and acute seneciosis in horses. Unfortunately the limited quantity of the alkaloids allowed only of one animal being placed in each of the two experiments.

Horse 21305 (8 months old): Received 2·0 gm, *isatidine* in a small quantity of moist bran by means of a balling-gun at 9 a.m. on the 10.3.36. As the animal developed no symptoms of poisoning it was given 5·0 gm. *retrorsine* on the 11.5.36. It remained in good health and was killed on the 12.6.36 for post-mortem purposes. There were general hyperplasia of lymphoid tissue including the spleen and dilatation of the stomach. A large number of Ascaris and Oxyuris worms were present. Microscopically no lesions were detectable in the liver, kidney myocard and spleen.

Horse 21304 (8 months old: Received 0·1 gm. *isatidine* daily in a moist bran by means of a balling-gun from 10.3.36 to 27.4.36 and 0·2 gm. *isatidine* from 28.4.36 to 16.5.36.

Also this animal remained in good health up to the time it was killed (26.6.36).

Autopsy revealed the following: Abscessation of the bronchial glands; slight acute catarrhal enteritis; extensive infestation with Ascaris, *Anaplocephala perfoliata*, and *Habronema microstoma*.

Also in this case no lesions were detectable upon microscopical examination of the internal organs.

In spite of the fact that negative results were achieved with the above experiments it would appear that *isatidine* and *retrorsine* are responsible, to a certain extent at least for the production of seneciosis as Davidson (1935) was able to produce typical lesions in the liver of rats with these alkaloids,

Our negative results may be due to (1) individual resistance on the part of the two experimental horses; (2) the feeding of too small amounts of *isatidine* and *retrorsine*. It is possible, and perhaps probable, that the two plants may contain other alkaloids chemically and toxicologically closely related to *isatidine* and *retrorsine*. According to the amount of plant material equivalent to the quantity of the two alkaloids administered both horses should have died; and (3) the two alkaloids having been stored for too long a period before being administered to the horses. This period was approximately twenty-two months.

This delay in the commencement of the experiment was unfortunately unavoidable as we had to collect certain information in regard to Senecio poisoning before the above experiment could be conducted.

It is felt that our negative results with the feeding of *isatidine* and *retrorsine* are possibly due to the alkaloids having become inactivated (changed) during the long period of storage. It should be stated that the alkaloids were stored in brown bottles in a dark cupboard.

HIPPOCRATEACEAE.

SALACIA REHMANNI SCHINZ.

Registered No.: O.P.H. No. 11780A; 18.2.36.

Common name: Wilde datel.

Origin: Middelfontein, Nylstroom, Transvaal.

State and stage of development: Wilted and in the late fruiting stage.

Rabbit A (2·4 Kg.): Received 10 gm. of the dry plant daily for three days.

Rabbit B (2·4 Kg.): Received 15 gm. of the dry plant daily for three days.

Result: Negative.

LEGUMINOCEAE.

CROTALARIA DURA WOOD AND EVANS.

Registered No.: O.P.H. No. 9569; 19.12.35.

Common names: Jaagsiektebossie, wilde lusern, wild lucerne.

Origin: The material was kindly collected in the vicinity of Pietermaritzburg, Natal, by Mr. R. A. Dyer, Botanist, Division of Plant Industry, Pretoria.

State and stage of development: Wilted and in the flowering and early seeding stage.

Horse 21303 (8 months old): Fed 1 Kg. of the dry plant mixed with lucerne hay and green feed (barley) daily from 10.3.36. The animal took the mixture readily.

Result.—27.3.36—Conjunctiva dark-reddish laboured respiration (deep and double expiration), losing in condition, apathetic.

28.3.36—As on previous day and not feeding well, fluid faeces.

From 29.3.36 to 26.4.36 the animal grew progressively worse.

27.4.36.—Staggering about in stable with wounds on all prominent parts of the body; conjunctiva swollen and yellowish in colour; pronounced laboured respiration; pulse weak and accelerated. The animal was killed in extremis.

Elevation of the temperature was recorded from the 21.3.36 to 24.3.36 (101·2-102° F.).

Approximately 20 Kg. of dry plant was eaten up to 29.3.36 at the rate of 1 Kg. daily. From this date up to the time of death the animal took very little food. The total amount of dry plant eaten is approximately 25 Kg.

Post-mortem Appearances.—Generalized abrasions; slight general icterus; emaciation; degenerative changes in the myocard; localised atelectasis; early pneumonic foci (?) in the lungs; regressive changes and cirrhosis of the liver; slight ascites; marked pigmentation and infarction of left kidney; slight acute mucocatarrhal enteritis; *Anaplocephala perfoliata*, gastrophilus larvae, Ascaris and Oxyuris plentiful.

Histology.—No pronounced lesions are detectable in the internal organs.

Crotalaria Globifera E. Mey.

Registered No.: O.P.H. No. 9568; 19.12.36.

Common names: Jaagsiektebossie, wilde lusern, wild lucerne.

Origin: Same as *Crotalaria dura.*

State and stage of development: Do.

Horse 21302 (8 months old): Fed 1 Kg. of the dry plant mixed with lucerne hay and green feed (barley) daily from 10.3.36 to 26.3.36. The total amount of dry plant eaten is 15 Kg. Unfortunately no more plant material was available.

Result.—At no time were any symptoms of poisoning discernible. The animal was killed on the 26.6.36 for post-mortem purposes. No macroscopic or microscopic lesions were detectable in the internal organs.

Owing to lack of knowledge of the difference in the botanical features of *Crotalaria dura* and *Crotalaria globifera* feeding experiments have in the past been conducted with mixtures of the two plants with the unfortunate result that *Crotalaria globifera* is also recorded to be toxic (Steyn, 1934). It is evident now that we have no experimental proof of the toxicity of *C. globifera*. It is possible that larger quantities of the plant than those fed to horse 21302 are required to produce poisoning.

LILIACEAE.

Ornithogalum Calcicola.

Registered No.: O.P.H. No. 183; 7.4.36.

Common name: —

Origin: Tsumeb, S.W.A.

State and stage of development: Fresh bulbs with no flowers or leaves

Rabbit (2·4 Kg.): Received 20 gm. of the fresh bulb at 11.30 a.m. on 7.4.36 and 40 gm. on the 8.4.36.

Result.—At 4 p.m. on 8.4.36 the animal was apathetic and purged profusely. It died with symptoms of paralysis, at 8 a.m. on 9.4.36.

Post-mortem appearances.—General cyanosis; hyperaemia of the lungs and liver; pronounced acute catarrhal gastro-enteritis with haemorrhages in the gastric mucosa.

ORNITHOGALUM CAUDATUM TIT.

Registered No.: O.P.H. No. 11175; 6.2.36.

Common names: —

Origin: Division of Botany, Pretoria.

State and stage of development: Fresh bulbs and leaves and in post-flowering stage.

Rabbit A (2·45 Kg.): Received 100 gm. of the fresh plant on each of two consecutive days.

Rabbit B (2·4 Kg.): Received 200 gm. of the fresh plant on each of two consecutive days.

Result.—Negative. These results confirm those obtained in previous experiments.

MYRISTICACEAE.

MYRISTICA FRAGRANS HOUTT.

Common names: Nutmeg; neut.

Rabbit A (2·3 Kg.): Received 10 gm. of ground nutmeg on each of two consecutive days.

Rabbit B (2·6 Kg.): Received 20 gm. of ground nutmeg on each of two consecutive days.

Result.—Negative.

Dog 1690 (18 months old, 4 Kg.): Received 5 gm. of ground nutmeg per stomach-tube daily from 28.5.36 to 5.6.36.

Result.—4.6.36—animal vomited 15 minutes after having been drenched; losing in condition.

5.6.36—Vomited after having been drenched; apathetic; not feeding.

The animal became progressively weaker and thinner and suffered repeated attacks of vomiting. It died during the night of 14.6.36.

Post-mortem appearances.—Hyperaemia of and degenerative changes in the liver; fibrosis of the kidneys; no food in stomach and intestines.

Dog 1845 (18 months old, 9 Kg.): Received 20 gm. of ground nutmeg per stomach-tube daily from 28.5.36 to 5.6.36.

Result.—Animal vomited after each dose from 30.5.36. It did not feed well and was losing in condition. It however recovered within a week after discontinuing the administration of nutmeg.

Nutmeg is sometimes used in cases of criminal abortion and has frequently caused poisoning [Lewin (1929), Stolte (1935)]. Lewin states that one nut may be sufficient to induce symptoms of poisoning. Stolte quotes a case of a child who had received 1 gm. of powdered nutmeg in his soup daily in the treatment of diabetes. This treatment had no effect on the diabetes but the child developed an itching urticarial exanthema.

PROTEACEAE.

GREVILLEA ROBUSTA CUNN.

Registered No.: O.P.H. No. 12981; 16.3.36.

Common name: Silver oak.

Origin: National Zoological Garden, Pretoria.

State and stage of development: Fresh leaves from a tree in the late seeding stage.

Sheep 40752 (Fullmouth, 37 Kg.): Received 500 gm. of the fresh leaves daily for five days.

Result.—Negative.

SANTALACEAE.

THESIUM TRIFLORUM THUNB.

Registered No.: O.P.H. No. 10987; 28.1.36.

Common name: Gifbossie.

Origin: " Onbekend ", Middelburg, C.P.

State and stage of development: Wilted and in fruiting stage.

Rabbit A (2·0 Kg.): Received 10 gm. of the dry plant on each of two consecutive days.

Rabbit B (2·45 Kg.): Received 15 gm. of the dry plant on each of two consecutive days.

Result.—Negative.

A second batch of this plant collected at the Grootfontein School of Agriculture, Middelburg, C.P., was dosed in the dry state and same stage of development to two rabbits with negative results.

UMBELLIFERAE.

FOENICULUM VULGARE MILL.

Registered No.: O.P.H. No. 3145; 11.7.36.

Common names: Vinkel, fennel.

Origin: Estcourt, Natal.

State and stage of development: Fresh plant with no flowers or seeds.

Rabbit (2·2 Kg.): Received 50 gm. of the fresh plant in one dose.

Result.—Negative.

VERBENACEAE.

VERBENA BONARIENSIS LINN.

Registered No.: O.P.H. No. 1196; 6.5.36.

Common names: —

Origin: Weenen, Natal.

State and stage of development: Almost dry and in the flowering stage.

Sheep 40752 (Fullmouth, 50 Kg.): Received 300 gm. of dry plant daily on four consecutive days.

Result.—Negative. No symptoms of photosensitisation seen.

ZYGOPHYLLACEAE.

ZYGOPHYLLUM MICROCARPUM LICHST.

Registered No.: O.P.H. No. 10432; 15.1.36.

Common names: Ou-ooibos, Armoedsbos, sandrepuis.

Origin: " Rooiberg Suid ", Maltahöhe, S.W.A.

State and stage of development: Fresh plant in the seeding stage.

Sheep 39750 (fullmouth, 50 Kg.): Received 600 gm. of the dry plant daily from 10.2.36 to 12.2.36 inclusive, and 300 gm. on 13.2.36.

Result.—11.2.36—slight hoven.

12.2.36—Fairly pronounced hoven; apathetic, not feeding well.

13.2.36—Pronounced hoven; dyspnoea; accelerated and strong heart-beat. Small quantity of ruminal contents flowing from nostrils.

14.2.36—Died previous night.

Post-mortem appearances.—Pronounced general cyanosis; abdomen markedly distended; subepicardial haemorrhages; hyperaemia of the lungs; liver and spleen decomposed.

SUMMARY.

Eighteen plants were tested biologically and one (*Dimorphotheca nudicaulis*) was tested chemically and found to contain a large quantity of hydrocyanic acid. In addition the effects of the alkaloids *isatidine* isolated from *Senecio isatideus*, and *retrorsine* isolated from *Senecio retrorsus*, were ascertained on horses. No positive results were however achieved.

Pergularia gariepensis and *Ornithogalum calcicola* were proved toxic. No previous records of their toxicity could be found in the literature consulted.

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Section VI.

Mineral Metabolism and Nutrition.

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Studies in Mineral Metabolism XXXVIII.

Calcium and Phosphorus in the Nutrition of Growing Pigs.

By SIR A. THEILER†, P. J. DU TOIT and A. I. MALAN, Section of Bio-chemistry and Nutrition, Onderstepoort.

BETHKE and associates (1933) and Dunlop (1935) conclude that growing pigs require approximately 0·6 per cent. P in their rations and that maximum growth is obtained with a Calcium-phosphorus ratio between 1:1 and 2:1. Bethke et al. furthermore state that as the proportion of Ca to P exceeded 3:1 the pigs became more rachitic and their vitamin D requirements increased. Our own experience here has been that while the ratio of Ca to P is undoubtedly important, osteodystrophic diseases may be produced in cattle, and probably in other species, merely by limiting the Ca or P intake or both, even when the ratio of these two constituents to one another was normal, when judged by the usually accepted standards; these observations have been summarized and discussed in the light of the findings of other investigators by Theiler and others (1936). Shohl and Wolbach (1936) report similar results with rats.

It would appear therefore that the relation between the ratio of Ca to P of a diet and the absolute intake of these two constituents is by no means settled, nor is it certain whether relatively high Ca with respect to P supplied in deficient amounts produces the same bone disease as the reverse, viz. high P and low Ca in the ration. Pigs were selected as the experimental animals as most of the more recent experiments at this Institute have been carried out on cattle and sheep, the results of which need obviously not apply to pigs.

Marek and his school (1932) have carried out experiments on Ca:P metabolism in pigs for the last twenty years and more and have expressed their conclusions in regard to the production of osteodystrophic disease subsequent to what they regard as abnormal Ca:P metabolism in pigs as follows:—

If the difference per 100 gms. dry feed consumed between Ca + Mg and P expressed as oxides does not fall within the comparatively narrow range of 20 and 25 mgm.-equivalents the development of osteodystrophic diseases follows. Magnesium should not exceed a third of the calcium content of the feed. It is not clear from Marek's work whether the above is generally true or only when vitamin D is present in deficient quantities. The above conception is clearly a modification of the Calcium-phosphorus ratio and does not make allowance for the presence of insufficient amounts of Ca and P when the difference referred to above, i.e. Erdalkali-Alkalizität (E.A.) of Marek, can be made to lie within the limits laid down by him. It must be admitted, however, that although an E.A. of 20-25 is regarded as normal by Marek he reports several cases of osteodystrophic disease in pigs on rations whose Ca, P, Mg contents gave normal values and again several animals remained healthy when the E.A. was distinctly abnormal. For instance pig No. 6 receiving daily per 10 Kg. body-weight on an average 3·435 gms. CaO and 5·591 P_2O_5 showing a strongly negative Erdalkali-Alkalizität remained healthy after 115 days in the experiment. Pig No. 27 received daily 3·576 gms. CaO and 1·9 gms. P_2O_5 per 10 Kg. bodyweight with an E.A. of +21·08 and developed severe rickets after 58 days in the experiment. Pigs No. 32, 33, 35, 39, 40 and 41 all remained healthy and with the exception of the last two received less than 1 gm. CaO per 10 Kg. liveweight but were given a supply of vitamin D by irradiation of the feed or in the feed; the E.A. ranged from slightly to strongly negative. Apparently the limits for E.A. hold good only when vitamin D is present in deficient amounts. If so, then it is not clear why pigs No. 27 and 28 should have developed severe rickets on an intake per 10 Kg. liveweight of approximately 3·5 gms. CaO and 1·8 gms. P_2O_5 with an E.A. of about 21 mgm. equivalents. In short, it would seem that, like the Ca : P ratio, Marek's Erdalkali-Alkalizität provides an inadequate basis for a discussion of the occurrence of osteodystrophic disease in pigs. If the intake of Ca or P, entirely apart from the ratio in which they occur or from the E.A. of the ration, can be made to determine whether or not bone disease develops, even if the animal can be made more sensitive to borderline intakes of Ca and P by altering the ratio, then surely the occurrence of osteodystrophic disease should invariably be related to the intake of Ca and P in the first instance unless of course the investigations are carried out under conditions of vitamin D deficiency when, generally speaking, the animal remains healthy within comparatively narrow limits of Ca : P intake, calcium phosphorus ratio or Erdalkali-Alkalizität. Abundant proof is available for the latter statement as a consideration of the work of Marek on E.A. and those of many investigators of Ca : P ratio will indicate (Shohl and Wohlbach 1936; Bethke and associates 1932, 2933; Dunlop 1935, etc.). But the occurrence of disease under conditions of vitamin D deficiency is the result of at least two factors, viz. abnormal ratio or intake of Ca and P or E.A. on the one hand and deficient vitamin D on the other. If however the latter factor be supplied the relation between abnormal Ca P metabolism and disease is a direct one which justifies investigation especially as this problem of abnormal Ca P metabolism, frequently caused by P deficiency, is unassociated with vitamin D deficiency in most sub-tropical countries.

An attempt was made in the preliminary experiments to be reported in this publication to study the effect on pigs of straight P or calcium deficiencies or both, while related factors such as E.A. and ratios of Ca to P naturally had to be considered.

EXPERIMENTAL DETAIL.

Uniform Large White piglets bred for experimental purposes at this Institute were selected from the available stock and divided into pairs. Individual feeding was practised for which purpose it was found best to run the pairs together and place each of a pair in a separate pen for feeding purposes from 12 noon until the following morning when the pair mates were let out into the adjoining common pen which was unprotected from weather conditions and hence afforded exposure of the pigs to the sun. No bedding was provided except for a short period before artificial heating of the piggeries had been installed when begasse or coarse fibrous sugar cane left after the cane sugar had been extracted, was supplied. For the rest a wooden board was placed in each pen on the concrete floor and the piglets generally accepted these as their beds without difficulty.

Water was always available in the common pen of each pair of piglets and by moistening the mash and feeding at maximum intake, but reducing feed whenever any was left over, food consumption was easily controlled and kept at maximum intake for each pair. All the pens were scrubbed and washed with water under pressure from a hose daily. This routine procedure was practised throughout the course of the experiments and left the animal undisturbed throughout the day and night except at feeding and washing time.

The piglets were weighed weekly, inspected daily for clinical symptoms of disease and blood was drawn and analysed for Ca, P and phosphatase at irregular intervals when technical assistance for the analyses was available; food consumption was recorded daily. Portions of the chondro-costal junction of the ribs were removed under anaesthesia from some of the piglets periodically for microscopical examination while others were killed at certain stages in the experiments for bone study—microscopical, physical and chemical. In some cases X-ray photographs were taken of the front legs of the animals which were placed under anaesthesia for this purpose.

EXPERIMENT I.

Ration Deficient in P but Adequate in Other Respects.

Pigs Nos. 987 and 993 aged approximately 6 months were given the following basal ration daily: 900 gms. maize-samp-meat meal mixture containing 10 per cent. of washed meat meal; 100 gms. green feed and 100 ml. fresh milk.

The meat meal contained approximately 80 per cent. protein and was washed twice in a very dilute HCl solution to reduce its mineral content and then with water several times to remove the acid. The washed product contained 0·1 per cent. P and ·06 per cent Ca. The milk was given after diluting it with 300 ml. of water and was immediately consumed. The samp-meat meal mixture was fed dry, while the green feed was placed alongside the trough on the concrete floor. $CaCO_3$ was added to the milk and water and well stirred in order to regulate the Ca intake according to the requirements of the experiment. As the appetite of the animals improved with growth the quantity of the samp-meat meal mixture was increased, the rest of the ration remaining constant.

The average daily intake of P and Ca throughout the course of the experiment was ·8 gms. and 6·0 gms. respectively with a ratio of Ca : P of 7·5 : 1.

The control animals, Nos. 989 and 990, were kept on the same basal ration except that unwashed meat meal was used instead of the washed product. Ca and P were added as $CaCO_3$ and Na_2HPO_4 to ensure an average daily intake of Ca and P of 6·0 gms. and 3·0 gms. respectively; Ca : P, 2·0 : 1.

The experiment began in January, 1934. In March Nos. 987 and 993 on the P-deficient ration began to show signs of poor appetite while the food consumption of the control pair continued to increase. In May five months after the beginning of the experiment, each pig of the control pair was consuming 1,800 gms. samp-meat meal mixture, while Nos. 987 and 993 ate daily only 600 gms. and at the conclusion of the experiment in October the daily feed consumption of the remaining control and remaining P-deficient animal was 2,400 and 400 gms. respectively.

Pigs Nos. 987 and 993 showed signs of stiffness from June onwards but apart from that and, naturally, poor growth and condition, both lasted well. No. 987 was killed for bone study on 1.9.1934 and No. 993 2½ months afterwards on 20.11.1934. A control animal was killed on each of the two dates for purposes of comparison.

The weekly body weights of the two pairs of pigs are given in Figure I.

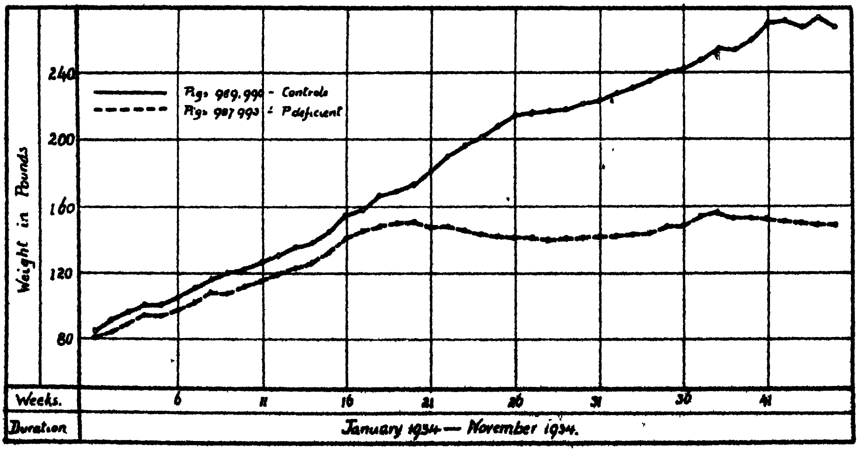

Fig. 1.

Weight increase appeared to be similar for the first five months of the experiment, i.e. until approximately 140 lb. was reached. From then onwards until the end of the experiment the pair on P-deficient diet showed no further increase, while the control pair continued to grow normally. The death of one pig in each group in September did not influence the increase in weight of the remaining pigs.

Inorganic phosphorus was determined in the blood in April, i.e. four months after the beginning of the experiment and the values obtained for the P-deficient group, viz. 3·6 and 3·7 mg P per 100 ml. blood suggest P deficiency while normal values of 8·8 and 9·0 mgm. were obtained for the two animals in the control group. The Ca content of the blood was determined only once during the course of the experiment, viz. in April when all the four values registered were approximately 10 mgm. Ca per 100 c.c. blood.

A femur of each pig was analysed at the end of the experiment and the results are shown in Table I.

TABLE I.—*On Green Weight.*

Nos.	Experimental details.	Green weight.	% fat and water.	% dry fat free bone.	Specific gravity.	% ash in dry fat free bone.
989	normal............	278·3	52·2	47·8	1·13	62·5
990	normal............	380·0	51·6	48·8	1·07	63·1
987	P. low............	236·7	63·5	36·3	0·97	57·4
993	P. low............	261·0	63·7	37·3	1·0	54·4

It will be noticed from the results given in the table that the quantity of bone material in the femurs of Nos. 987 and 993 is considerably less than that in the bones of the control group, viz. 36·8 per cent. as against 48·1 per cent. Furthermore the percentage ash, calculated on the fat free bone weight is only 55·7 in the femurs of the P-deficient group and 62·8 in the control group. There can be no doubt that the bones of the former group were abnormal.

Sections of the ribs were examined microscopically and both animals were found to show rachitic lesions. Pig No. 993 which remained almost three months longer in the experiment than No. 987 showed lesions of marked rickets while slight rickets was diagnosed in the case of the group mate No. 987.

From a consideration of the data presented there can be no doubt that the ·8 gm. P supplied in the rations of pigs Nos. 987 and 993 was insufficient for normal growth. Increase in weight took place for the first 5 months of the experiment while the skeletal reserves of the pigs lasted and thereafter the bodyweight remained practically constant. Not only was growth absent but rachitic lesions developed due to the phosphorus deficiency. It is surprising that the animals lasted the full time of the experiment, which was probably due to the fact that they were 6 months old at the beginning and had built up a considerable mineral reserve in their skeleton. Hence it was decided to determine the effect of P deficiency on younger pigs.

Four 8-weeks-old piglets were accordingly selected. A difference in the method of feeding was introduced into this trial; the minerals were added to the samp of the group and mixed in bulk at the beginning of the experiment and were not fed in the milk. It was also thought advisable to introduce the vitamin D factor into this experiment. Hence in every group one pair of piglets was kept in semi-darkness and the other pair on the same ration but given free access to light.

The four control piglets Nos. 1133, 1143, 1138 and 1140 of which the latter two were kept in semi-darkness were given the following ration:—

100 ml. milk.
100 gms. green feed.
Mash according to appetite.

The mash consisted of—

90 parts maize samp
10 parts high protein meat meal,
2·5 parts $Na_2 HPO_4$ (19 per cent. P).
2·2 parts $CaCO_3$ (40 per cent. CaO).
1 part NaCl.

The Ca and P content of the mash was 1·0 and ·55 per cent. respectively.

Piglets Nos. 1135, 1144, 1131 and 1137 of which the latter two were kept in semi-darkness were given essentially the same rations as the controls, but the P intake was reduced to a minimum by omitting the $Na_2 HPO_4$ from the ration and the effect of the P deficiency so created was made more severe by increasing the $CaCO_3$. The ration consisted of the following:—

100 ml. milk.

100 gms. green feed.

The mash which was fed according to appetite consisted of—

90 parts maize samp.

10 parts high protein meat meal.

4·9 gms. $CaCO_3$.

1 part NaCl.

The P and Ca content of the mash was ·709 and 2·0 per cent. respectively and the milk and green feed which were given contained ·21 gms. Ca and ·152 gms. P.

The darkness in the pens of the pigs kept in the dark was so intense that the troughs, etc., could be seen indistinctly and only after accustoming one's eyes to the darkness.

The experiment began in November, 1935; one animal in each group was killed in February, 1936, for bone studies.

A portion of the costo-chondral junction of a rib of the remaining group mates of the P-deficient group was removed in March and again in April, after which P was supplied and rib resections repeated on all the group mates in June when the experiment was discontinued.

Data in regard to the body weights are supplied in the following figures.

Figure 2 represents the comparison of the body weights of the pair of control pigs kept in darkness throughout the experiments and the pair given free access to light.

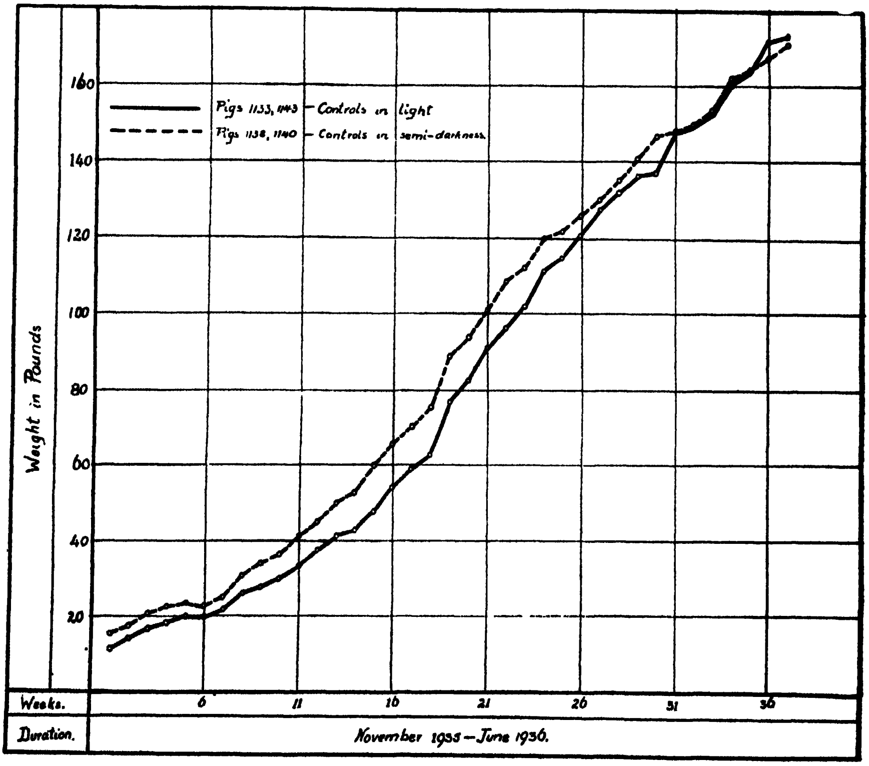

Fig. 2.

It is remarkable that after two months no significant difference existed in the body weight of the two pairs of pigs and that even after eight months the remaining pigs of each pair still showed practically the same increase in body weight. The increase in body weight undoubtedly suggests that the vitamin D content of the feeds used is sufficient for the normal growth of pigs for at least eight months when given adequate rations. This finding is in agreement with that of Huffman et al. (1935) with dairy calves given an adequate supply of hay. It would be interesting to know whether pigs may be reared to maturity practically in the absence of light and further work has been undertaken along these lines.

The body weights of the two pairs of pigs on the P-deficient diet are compared in Figure 3.

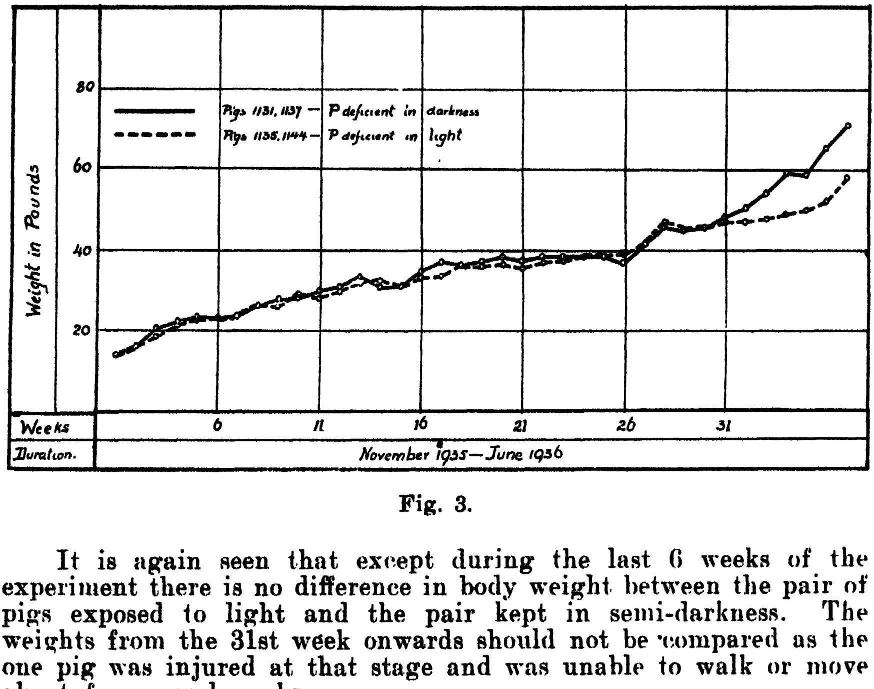

Fig. 3.

It is again seen that except during the last 6 weeks of the experiment there is no difference in body weight between the pair of pigs exposed to light and the pair kept in semi-darkness. The weights from the 31st week onwards should not be compared as the one pig was injured at that stage and was unable to walk or move about for several weeks.

It appears, therefore, that the Ca and P under conditions of excess of the former and a deficiency of the latter were equally well utilized whether or not the pigs had access to light. The ration apparently contained sufficient vitamin D for the requirements of the pigs.

In Figure 4 the average body weight of the 4 pigs of the control group is compared with that of all the 4 pigs on the P-deficient ration and the curves show a remarkable contrast, especially if the latter portion of the curves, i.e. from the 26th week onwards when P was given to the P-deficient group be not considered.

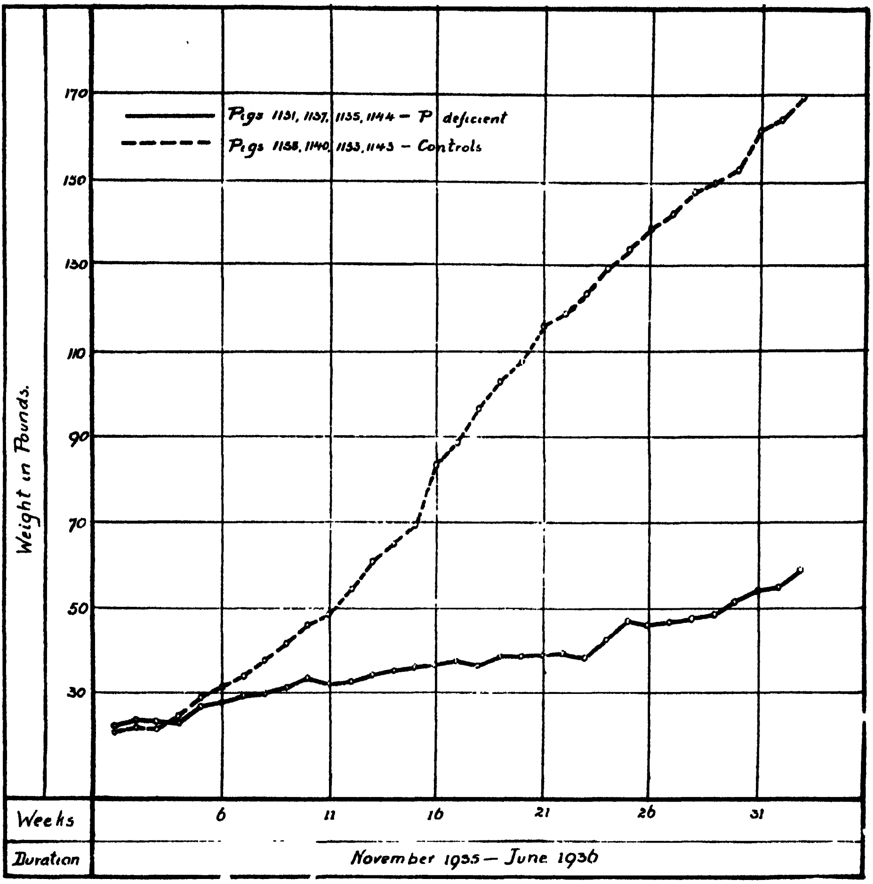

Fig. 4.

It is to be noted that the pigs receiving a P-deficient ration showed a total increase in body weight of only about 20 lb. during the 26 weeks experimental period as against 110 lb. for the control group. Growth in the P-deficient group was almost negligible which was also indicated by the poor food consumption and condition.

The average food consumption per pig per day calculated for the separate months is given in Table II.

TABLE II.

Weights given in gms.

No. of Pig.	Ration.	Nov.	Dec.	Jan.	Feb.	March.	April.	May.	June.
1138, 1140, 1133 and 1143	**Controls in sun and darkness**	**370**	**750**	**1,100**	**1,300**	**1,500**	**1,600**	**1,700**	**1,750**
1131, 1137, 1135 and 1144	**P deficient in sun and darkness**	**400**	**535**	**720**	**460**	**350**	**720**	**740**	**1,000**

TABLE III.

Phosphatase (Bodansky units) and Inorganic Phosphorus (mgms. per 100 ml. serum).

No. of Pig.	Experimental detail.	7/11/35.		4/12/35.		21/1/36.		11/2/36.		23/3/36.		5/5/36.	
		Ph.	IP.	Ph.	IP.	Ph.	IP.	Ph.	IP.	Ph.	IP.	Ph.	IP.
1138	Control pig in darkness	7·2	8·7	5·3	8·0	7·6	9·0	7·0	8·1	9·4	8·0	10·0	8·2
1133	Control pig in light	7·4	8·0	10·7	7·8	12·2	9·2	—	7·8	11·8	8·6	12·1	9·1
1131	P deficient pig in darkness	6·9	7·5	—	6·4	17·5	4·2	18·0	3·0	—	—	—	10·6
1135	P deficient pig in light	—	—	—	—	17·2	3·3	—	—	—	—	9·9	9·1

TABLE IV.

Analysis of femur.

No. of pig.	Experimental details.	Green weight. Gms.	On green weight.		On dry fat free bone.			On ash.		
			% fat and water.	% dry fat free bone.	% ash.	% Ca.	% P.	% Ca.	% P.	Ca : P.
1133	Ca+P+D−	100·1	57·6	42·4	62·3	23·7	11·6	38·1	18·6	2·05 : 1
1140	Ca+P+D−	106·2	58·2	41·8	61·4	22·9	11·3	37·3	18·4	2·03 : 1
1131	Ca+P−D+	63·4	60·1	39·9	48·9	17·7	9·0	36·2	18·4	1·97 : 1

Food consumption in the P-deficient group decreased continually until March when P was added to the ration and food intake more than doubled itself during the following month. During March, i.e. the month of poorest consumption, the control pigs ate more than four times the quantity of mash consumed by the P-deficient pigs.

Phosphatase and the inorganic phosphorus content of the serum were determined six times during the course of the experiment and the results are submitted in Table III.

The inorganic P content of the serum confirms P deficiency which was apparently most severe in February. The last values for Nos. 1131 and 1135 determined after P had been given for several months indicates a sufficiency of P in the ration. In the control group P sufficiency is suggested by the values of P throughout the experiment. It is noteworthy that the phosphatase values of the control pigs kept in the dark appear to have been consistently lower for several months of the experiment than those of the pigs receiving the same feed but allowed free access to sunlight. The normal values for phosphatase were less than 10 Bodansky units and the values of the pigs suffering from P deficiency increased to approximately 17 and were reduced to normal values after the addition of P in March, 1936.

Calcium was not determined except once in the blood of the pig killed in each group during February. The values all range from 9·4 to 10·2 mgm. per 100 c.c. blood and show no group differences. For the phosphatase determinations blood was drawn from the caudal vein and it proved to be difficult to obtain enough blood for both phosphatase and calcium.

A femur of each pig killed in February was taken for analyses and the results are tabulated in Table IV.

Unfortunately the femur of No. 1135 on a P-deficient diet and kept in the dark was not kept for analysis which reduces the value of the analyses given very considerably. It would seem, however, that a marked difference existed between the femur of the control pigs when compared with that of the P-deficient pig. The percentage bone material was less and so was the ash when P was present in insufficient amounts. In regard to the P and Ca content of the ash and the ratio in which they occur more will be said at a later stage in this article when more data can be discussed.

From a consideration of the data given one fact stands out clearly, viz. that P deficiency affected the pigs detrimentally although the extent to which that happened does not seem to have been influenced by the presence or absence of sunlight. It cannot be said that the control pigs were affected with regard to any of the observations made by the presence or absence of light. It would be interesting therefore to mention how the pigs reacted clinically to the conditions of the experiment.

The experiment, as stated, was begun in November, 1935, and except for the poor appetites displayed by the P-deficient groups no difference was noticed between the P-deficient groups and the controls until the end of December. Naturally the former groups appeared less satisfied and less lively than the latter. During the first week of January both the P-deficient groups appeared to be less inclined to move about and even suggested stiffness.

The hocks seemed to sag as if the weights of the bodies were too great to be held up properly by the legs. Towards the end of January No. 1137 on the P-deficient diet in the dark was slightly but decidedly stiff. On 21.1.36 this animal seemed to be in pain and so disinclined to walk or stand that it sat on its haunches most of the time. Two days afterwards all four pigs on the P-deficient ration were decidedly stiff, showed disinclination to walk and showed sagging of the hocks. No. 1135 died during the night of 24.1.36 after consuming most of its food. The cause of death did not appear to be associated with P deficiency. The remaining three pigs on the P-low diet (1131 and 1137 in dark and 1144 in light) gradually became worse. From 10.2.36 onwards rising was a supreme effort and was preceded by hard struggling. The hind legs continually slipped under their bodies when rising. On the 21.2.36 when No. 1131 was killed for bone studies all three were very weak in their hindquarters. The thickened joints which had gradually become more pronounced during the last month were very noticeable. The pigs moved from side to side when walking and were weak.

The remaining two pigs, Nos. 1137 and 1144, were given the P-deficient ration for another month, during which period they remained practically constant in weight, underfed and in great pain when forced to move. A portion of the costo-chondral junction of the 6th rib was removed from each pig on 27.3.36 after which they were given a daily supplement of Na_2HPO_4 when they showed an immediate improvement. Ribs were again removed for microscopical examination on the 3/4, 17/4 and 10/6. The animals were then discharged.

During the whole of the experimental period the control animals remained normal. No clinical symptoms were noticed at any stage and the pigs remained lively and healthy. No. 1140 (in dark) and 1133 (in light) were killed for bone study on 21.2.36 and ribs removed from the remaining controls on 10.6.36 at the end of the experiment.

From an examination of the histological sections of the ribs, bone formation was proceeding normally in Nos. 1140 and 1133 when they were killed in February, as was also the case with 1138 and 1143 in June when the resected ribs were examined. In contrast with these findings the rib sections of the pig No. 1131 on the P-deficient ration when killed at the end of February showed abundant microscopic lesions of marked rickets. The bones were soft and easily fractured by applying moderate pressure. The rib of No. 1135 which died in January also showed severe rickets. No. 1137 whose rib was removed in March suffered from florid rickets at that stage—the trabeculae consisting almost entirely of a mass of osteoid. After Na_2HPO_4 had been given to Nos. 1137 and 1144 for 7 days rib sections were again examined and still showed rickets but less severe lesions than those of the previous week. Even after a fortnight a diagnosis of rickets could still be made although the animals were moving about freely and showed very marked improvement. However, they did not seem to be recovering and in June, 1936, three months after the phosphate supplement had been given they appeared stunted and the legs still decidedly abnormal although microscopical bone sections showed normal bone formation and certainly no suggestion of rickets. They moved about freely and easily and their appetites were excellent.

EXPERIMENT II.

Ration Deficient in Ca but Adequate in Other Respects.

Pigs Nos. 983 and 978 aged approximately 6 months, like the first pair described in Experiment I, were used and given the same basal ration as in that case. The treatment was the same and both experiments were conducted concurrently.

The daily Ca intake increased during the course of the experiment from 0·45 gms. at the beginning in January, 1934, when food consumption was low to 1·2 in May when almost 2 Kg. of mash was consumed per head daily and never at any time exceeding this point until the conclusion of the experiment in November, 1934. The ratio of Ca : P was kept constant at 1 : 10.

Nos. 989 and 990 receiving on an average 6·0 gms. Ca and 3 gms. P daily as described in Experiment I, are regarded as the controls of the pigs in this experiment and the results will be presented accordingly.

During the second month of the experiment both pigs on the Ca-low ration (Nos. 983 and 978) appeared to be slightly stiff in the hindquarters. The quantity of feed consumed daily increased from 900 gms. at the beginning of the experiment in January to 1,700 gms. in April and then gradually decreased to 800 in August and showed a slight increase towards the end.

In August No. 978 showed digestive disturbances and refused its feed altogether after a week. It died on 27.8.34. The experimental mate lasted until the end of the experiment.

The weekly body weights are presented in Figure 5.

The average body weight of the group receiving low Ca did not differ significantly from that of the control group during the first 7 months of the experiment but after that period the difference in weight between the groups became more pronounced. Still, the Ca-low group continued to increase gradually in weight.

Blood analysis for Ca and P revealed no difference between the groups in March when the determinations were made.

The results of the analysis of the femur of pig No. 983 is given in Table V. The control pigs are included in the table for comparison; the femur of No. 978 was not analysed as this pig died during the course of the experiment and the results would therefore not be comparative.

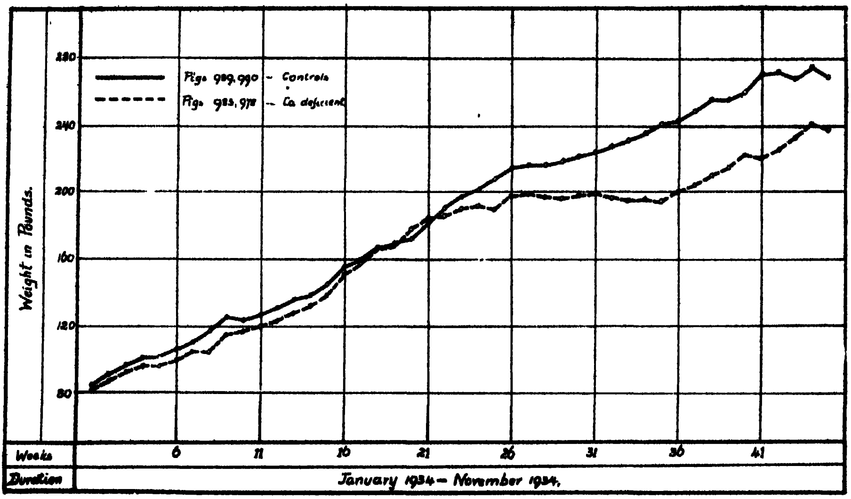

Fig. 5.

TABLE V.

On Green Weight.

Nos.	Experimental details.	Green weight.	% fat and water.	% dry fat free bone.	Specific gravity.	% ash in dry fat free bone.
989	Normal............	278·3	52·2	47·8	1·13	62·5
990	Normal............	380·0	51·6	48·8	1·07	63·1
983	Low Ca............	267·5	63·1	36·9	1·00	59·9

It would seem from the values given that the femur of No. 983 receiving low Ca in its diet contained less bone material than the femur of the controls but the number of determinations is too limited to justify any serious consideration of the values.

Sections of the ribs of both animals were examined microscopically. The sections of No. 978 which had suffered from digestive disturbances prior to death showed extensive lesions of esteoporosis and atrophy and No. 983 osteoporosis and slight atrophy. The important fact of the diagnosis was that neither pig showed rickets microscopically and that the bones were not normal.

Summarizing the conclusion that the quantity of Ca present in the diet viz., approximately 1 gram per daily ration was insufficient for normal growth and bone formation, is justified. Apparently growth continued normally for the first 6 months of the experiment and only after that period could the skeletal reserves of lime no longer supply the body requirements of calcium, when growth began to suffer. The pigs were 6 months old at the beginning of the experiment which may account for the fact that they lasted well in spite of their low Ca intake. It was therefore decided to determine the effect of a low Ca intake upon younger pigs.

Four eight-weeks-old pigs were therefore selected and placed on the following daily ration:—

100 ml. milk.
100 gms. green feed.
Mash according to appetite.

The mash consisted of—

90 parts maize samp,
10 parts high protein meat meal.
5·8 parts Na_2HPO_4 (40 per cent. P_2O_5), and
1 part NaCl.

The P and Ca content of the mash was ·98 and ·11 per cent. respectively, while the daily milk and green feed supplied ·21 gms. Ca and ·152 gms. P.

This experiment was conducted concurrently with the second part of Experiment I and the same four animals, viz. No. 1138, 1140, 1133 and 1143 acted as controls to both experiments. As already stated all four animals were given the same normal ration and the only difference in management was that the latter two animals were allowed free access to direct sunlight when they were not feeding while the two former ones were always in semi-darkness.

The pigs receiving low Ca in their diets were also divided into two pairs. The one pair, viz. Nos. 1130 and 1145 was kept in semi-darkness whereas Nos. 1136 and 1142, receiving the same ration and treatment, were given free access to direct sunlight.

The experiment began in November, 1935, and continued for approximately eight months.

The average body weight of Nos. 1130 and 1145 on low Ca and in darkness is compared graphically in Figure 6 with that of Nos. 1136 and 1142 which had received the same ration but were allowed free access to light.

The body weights of the two pairs of pigs did not differ significantly during the first 22 weeks of the experiment, i.e. until March, 1935. Nos. 1136 and 1145 were killed for bone studies at the end of February and the remaining pig No. 1130 which was allowed free access to light developed severe diarrhoea shortly afterwards from which it suffered acutely for approximately two months and then died. Hence the body weights are not comparable after the 22nd week of the experiment. Prior to that period it seems that the body weights of the pigs kept on a Ca-deficient diet were not affected by the presence or absence of light.

The average body weight of pigs Nos. 1130 and 1145 on low Ca but kept in semi-darkness is compared with that of the corresponding control pair in Figure 7.

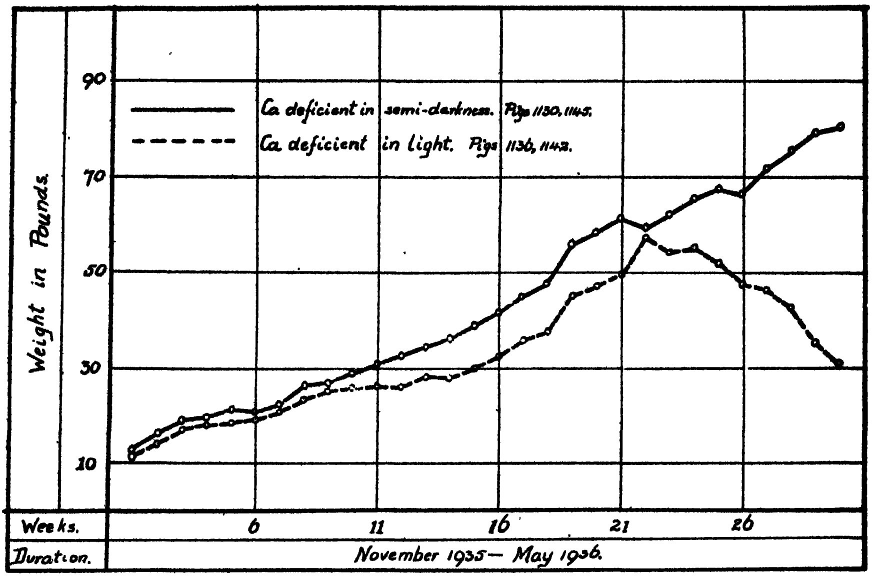
Weight in Pounds.
90
70
50
30
10
Ca deficient in semi-darkness. Pigs 1130, 1145.
Ca deficient in light. Pigs 1136, 1142.
Weeks.
6
11
16
21
26
Duration.
November 1935— May 1936.

Fig. 6.

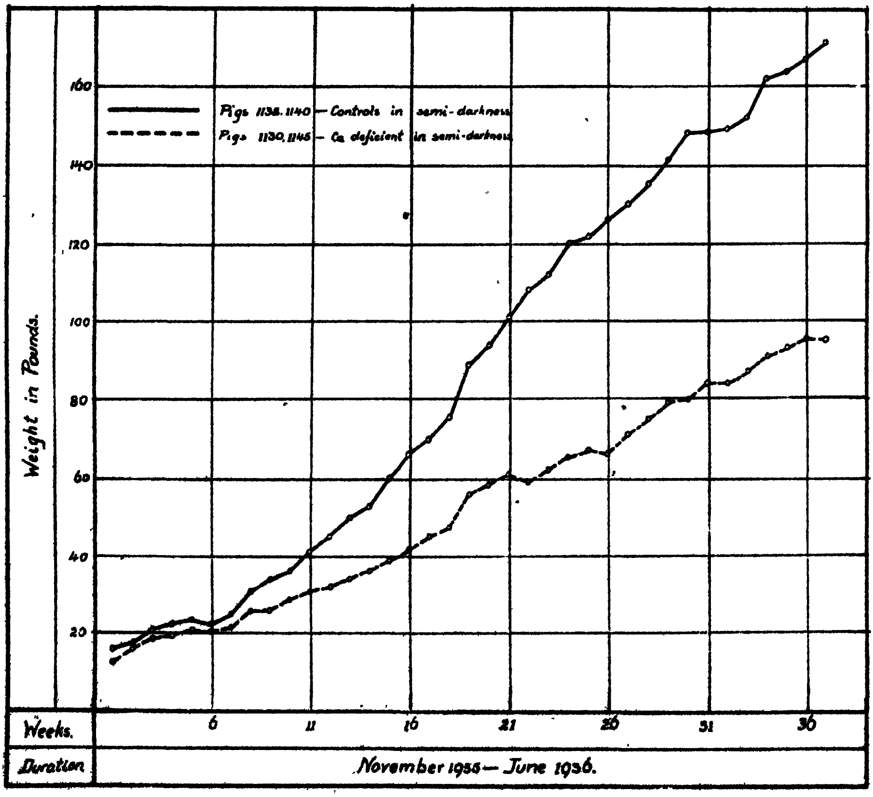
Weight in Pounds.
160
140
120
100
80
60
40
20
Pigs 1138, 1140 — Controls in semi-darkness
Pigs 1130, 1145 — Ca deficient in semi-darkness
Weeks.
6
11
16
21
26
31
36
Duration.
November 1935— June 1936.

Fig. 7.

Low Ca in the diet affected the body weight almost from the beginning of the experiment and was responsible for the difference of about 70 lb. at its conclusion eight months afterwards. In spite of a low Ca intake, however, a gradual increase in weight continued throughout the course of the experiment; there was no cessation of growth as in the case of the P-deficient pigs.

The average food consumption, which is given in Table VI, of the pigs on a Ca-low diet compared with that of the controls confirms the observations made in regard to the increase in body weight.

TABLE VI.

Daily Intake of Mash per Pig: Weights given in gms.

No. of Pig.	Ration.	Nov.	Dec.	Jan.	Feb.	March.	April.	May.	June.
1138, 1140, 1133 and 1143	Controls in sun and semi-darkness	370	750	1,100	1,300	1,500	1,600	1,700	1,750
1130, 1145, 1136 and 1142	Ca deficient diet in sun and semi-darkness	370	470	680	1,000	750	900	900	1,046

Food consumption was affected detrimentally by low Ca in the diet and more so as the experiment continued.

The values of phosphatase and the inorganic phosphorus content of the serum are given in Table 7.

It seems that neither phosphatase nor the inorganic phosphorus content of the serum was affected by the Ca content of the ration but the number of determinations is too limited to justify any conclusions being drawn in regard to phosphatase.

Calcium determined in the blood of the pigs of each pair that was killed in February revealed no change and was still apparently normal.

The femurs of the two pigs killed in February were analysed and the results presented in Table VIII together with the values of the control pigs.

The percentage bone material was considerably reduced in the green bone of the animals receiving low Ca in their rations. The ash content of the dry fat free bone was also significantly lower than that of the normal bones, although the percentage Ca and P of the ash remained practically constant.

Rib sections of the two pigs on a low Ca diet Nos. 1145 and 1136 killed in February, four months after the beginning of the experiment, were examined microscopically and showed osteoporosis but no lesions of rickets. It will be remembered that No. 1145 was kept in semi-darkness during the experiment and that No. 1136

TABLE VII.

Phosphatase (Bodansky Units) and Inorganic Phosphorus (I.P.) in mgms. per 100 *c.c. Serum.*

No. of Pig.	Experimental detail.	7/11/35.		4/12/35.		21/1/36.		11/2/36.		23/3/36.		5/5/36.	
		Ph.	I.P.	Ph.	I.P.	Ph.	I.P.	Ph.	I.P.	Ph.	I.P.	Ph.	I.P.
1138	Control pig in darkness	7·2	8·7	5·3	8·0	7·6	9·0	7·0	8·1	9·4	8·0	10·0	8·2
1133	Control pig in light	7·4	8·0	10·7	7·8	12·2	9·2	—	7·8	11·8	8·6	12·1	9·1
1145	Ca def. pig in darkness	6·8	9·2	—	—	8·0	6·5	—	10·3	—	—	—	11·1
1142	Ca def. pig in light	7·7	7·7	9·1	8·2	8·2	6·3	4·9	10·6	—	—	—	7·6

TABLE VIII.

No. of pig.	Experimental details.	Green weight. Gms.	On green weight.		On dry fat-free bone.			On ash.		
			% fat and water.	% dry fat free bone.	% ash.	% Ca.	% P.	% Ca.	% P.	Ca : P.
1140	Control in darkness.	106·2	58·2	41·8	61·4	22·9	11·3	37·3	18·4	2·03 : 1
1133	Control in light	100·1	57·6	42·4	62·3	23·7	11·6	38·1	18·6	2·05 : 1
1145	Ca low in darkness	76·8	68·6	31·4	55·8	20·8	10·3	37·3	18·5	2·02 : 1
1136	Ca low in light	57·0	69·4	30·7	55·3	19·9	10·4	36·0	18·8	1·96 : 1

had free access to light. Ribs were removed from the two remaining pigs on low Ca in March and again in June at the conclusion of the experiment. The sections of the ribs of both pigs showed marked bone atrophy or osteoporosis but no other lesions of osteodystrophic disease.

From a consideration of the data presented it is clear that ·11 per cent. of Ca in the mash of growing pigs was insufficient for normal growth and bone formation when 2·25 per cent. of P was present. It seems, however, that the deficiency was not acute enough to produce clinical symptoms of bone disease during the eight months that the experiment lasted. None of the four pigs on the Ca-low diet whether free access was given to light or not, showed signs of stiffness or any clinical symptoms which could be associated with calcium deficiency. The ration consisted of 100 ml. milk and 100 gms. green feed daily in addition to the mash.

A pair of pigs of the same age, viz, eight weeks, as those used in Experiment 5 was placed on a calcium-low ration when the latter experiment was conducted and the results obtained might briefly be reported here.

The daily ration given consisted of 100 ml. milk, 100 gms. green feed and mash consisting of 94 parts maize samp, 6 parts blood meal and 1 part of salt according to appetite. Na_2HPO_4 was added daily to the ration to ensure an intake of ·5 gms. CaO, 10·5 gms. P_2O_5 and a ratio of 1:21.

Both pigs (Nos. 1075 and 1089) showed poor appetite almost from the beginning of the experiment. No. 1089 developed severe diarrhoea and died of acute enteritis shortly after the beginning of the experiment. No. 1075 showed no digestive disturbances but did not relish its food. Approximately two months after the beginning of the experiment this pig showed unmistakable symptoms of stiffness and weakness. The hindquarters were apparently unable to support the body and especially on turning the pig would go down on its haunches and remain in that position for a few minutes. The pig appeared to improve slightly during the following week but on being driven to the scale for weighing it suddenly became lame and was quite unable to rise for several days. It ate its food in a lying position and was unable to change its position without assistance. During the remaining six weeks in the experiment this pig on several occasions developed lameness and subsequent inability to move around. It was always stiff when able to move about. The pig was killed on 13.8.35, i.e. after 105 days in the experiment for histological study of the bones. Numerous callusses were present in the ribs and the bones were obviously soft and brittle. Histological sections of the ribs showed marked atrophy and incipient osteodystrophia fibrosa but no rickets. The radiographs taken after the pig had been in the experiment for about two months also showed marked atrophy.

Only one pig was left on the Ca-low diet and the development of incipient osteofibrosis cannot therefore be ascribed definitely to low Ca but in the light of the results obtained with cattle, sheep and goats on Ca-deficient diets the possibility of a Ca-low diet leading to osteodystrophia fibrosa in pigs is tentatively suggested. Further work with pigs along these lines will be reported at a later stage.

EXPERIMENT III.

This Experiment was conducted concurrently with Experiments I and II and was an Attempt to Determine the Effects of a Combined Deficiency of Ca. and P.

It was realized that this experiment would throw further light on the effect of the ratio of Ca:P of the rations used in the first two experiments. Hence the P and Ca intakes were made to agree with those of Experiments I and II as the table given below indicates. A comprehensive table giving full details of all the experiments mentioned in this report is given on page 161.

Experiment.	Ca intake.	P intake.	Ratio Ca : P.
I. (6 months old pigs)..............	6 gms.........	·6 gms........	10 : 1
I. (8 weeks old pigs)...............	2 per cent......	·11 per cent.....	10·9 : 1
II. (6 months old pigs)..............	1 gm..........	10 gms........	1 : 10
II. (8 weeks old pigs)...............	·11 per cent.....	·98 per cent.....	1 : 8·9
III. (6 months old pigs)..............	1 gm..........	·6 gm..........	1·7 : 1
III. (8 weeks old pigs)...............	·11 per cent.....	·11 per cent.....	1 : 1

It must be noted that, although the ratio of Ca:P in Experiment III was more favourable than that of Experiment I or II a double deficiency existed in Experiment III.

Two six-months-old pigs Nos. 988 and 994 were given the same basal ration as that used for the pigs of this age in Experiments I and II, viz.:—

100 gms. green feed.

100 ml, milk.

Maize samp plus meatmeal mixed in the proportions of 90 parts to 10 respectively.

The experiment began in January, 1934, and both animals lasted the full period of the experiment, i.e. until November, 1936. In February No. 988 appeared to be slightly stiff in the front legs for a short period and again showed slight stiffness in August in the hindquarters. Other clinical symptoms were not noticed at any stage in the course of the experiment.

The average body weight of pigs Nos. 988 and 994 is compared graphically with that of the control pair in Figure 8.

It is evident from the graphs that the body weight of the pair of pigs receiving low Ca and P in their diet was practically identical with that of the control pair for the first twenty weeks of the experiment. After that period a difference set in which reached about 50 lb. at the end of the experiment in favour of the control pair.

The inorganic phosphorus content of the blood was found to be 5·7 mg. per 100 ml. which is lower than the control value but appreciably higher than that obtained for the blood of the pigs on the P-deficient ration and excess Ca. Blood Ca was normal.

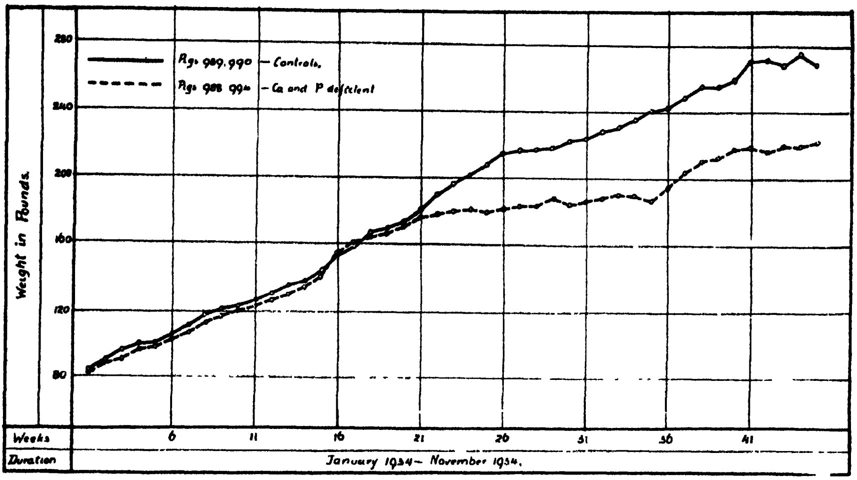

Fig. 8.

The results of the analyses of the femur are given in Table IX.

TABLE IX.

On Green Weight.

Nos.	Experimental details.	Green weight. Gms.	% fat and water.	% dry fat free bone.	Specific gravity.	% ash in dry fat free bone.
989	Normal............	278·3	52·2	47·8	1·13	62·5
990	Normal............	380·0	51·6	48·8	1·07	63·1
988	Low Ca and Low P.	248·8	58·1	41·9	1·00	57·8
994	Low Ca and Low P.	285·0	60·1	39·9	1·02	60·1

The percentage quantity of bone material on the femurs of Nos. 988 and 994 is less than that in the femurs of the controls which would indicate poorer bone formation; as a result the ash percentage of the former pair is also lower.

The rib sections of the Ca and P deficient pair of pigs showed microscopic lesions of extensive osteoporosis and indications of slight rickets; these facts corroborate the conclusions drawn from the bone analyses.

As already stated these pigs were approximately six months old when the experiment started and it was therefore decided to determine the effect of a ration low in both Ca and P on younger pigs.

Accordingly four eight-weeks-old pigs were selected and given the same basal ration as that of the pigs of the same age in Experiments I and II, viz.:—

100 ml. milk.
100 gms. green feed.
Mash according to appetite.

The mash was composed of 90 parts maize samp, 10 parts of meatmeal high in protein and 1 part of salt.

The Ca and P content of the mash was the same, viz. ·11 per cent. No extra mineral supplement was given.

Pigs Nos. 1132 and 1141 were given free access to sunlight, whereas Nos. 1139 and 1129 were kept in semi-darkness.

The comparative weight curves of the two pairs of pigs are given in Figure 9.

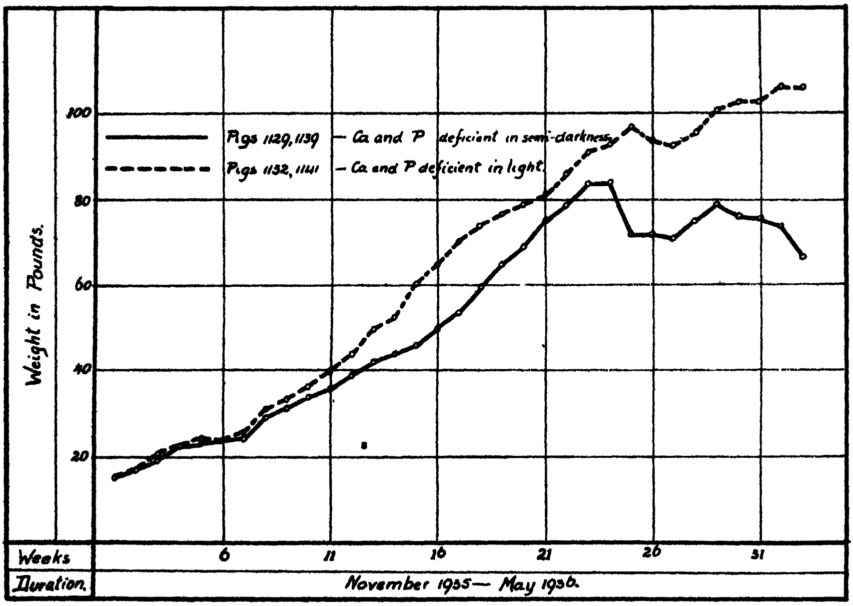

Fig. 9.

It is doubtful whether light had any effect on the body weights of the pigs before March, 1936, i.e. about five months after the beginning of the experiment. One pig of each pair was killed for bone studies towards the end of February and it was after that period that the body weights of the remaining pigs began to differ considerably. No. 1139 kept in the dark actually lost 20 lb. during the following nine weeks, while No. 1141, which was allowed free access to sunlight increased about 20 lb.

It would seem that the effect of the absence of light was being felt during this period. However, as only two pigs could be compared this conclusion must be drawn with reservation until further work has been done.

If the body weight of the pair of pigs Nos. 1132 and 1141 be compared with that of the control pair which was also allowed access to sunlight it is seen that low Ca and P began to affect the body weights of the pigs adversely about twenty weeks after the beginning of the experiment. Weight increase was poor after that period and a difference of 70 lb. was registered between the two remaining pigs at the conclusion of the experiment. (Fig. 10.)

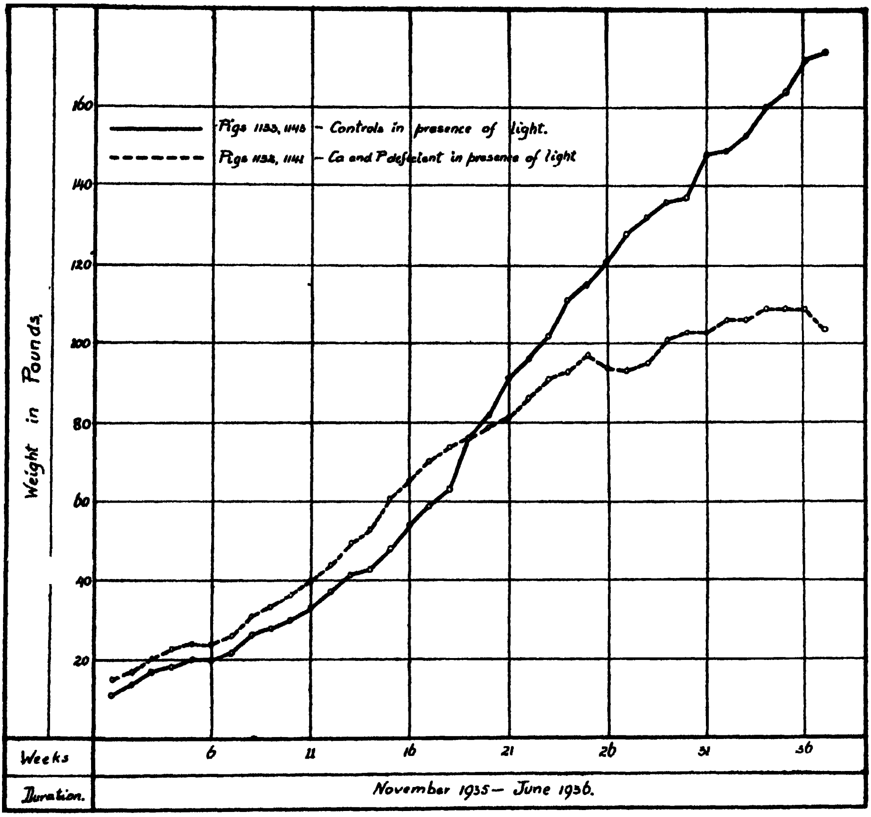

Fig. 10.

It is well to remember that the increase in weight of the pigs receiving a diet low in both Ca and P was by no means as poor as that registered in Experiment I, when P was deficient and Ca present in excess or as that given in Experiment II when Ca was deficient and P present in excess. The explanation of these apparently contradictory results, viz. that a deficiency of both Ca and P should produce better growth than a deficiency of either Ca or P, lies, of course, in the generally accepted view that the beneficial ratio in the first instance reduced the effects of the deficiencies while the abnormal ratio when either Ca or P was present in deficient amounts aggravated the effects of the deficiency to such an extent that it was actually more severely felt than when both elements were deficient.

Food consumption like the body weights shows reduction when compared with that of the controls but not to the same extent as when either Ca or P was present in insufficient amounts.

The average food consumption per pig per day during the separate months is given in Table X.

TABLE X.

Weights, Green in gms.

No. of Pig.	Ration.	Nov.	Dec.	Jan.	Feb.	March.	April.	May.	June.
1138, 1140, 1133 and 1143	Controls in darkness and sunlight	370	750	1,100	1,300	1,500	1,600	1,700	1,750
1132 and 1141	Ca and P deficient in light	370	713	1,080	1,200	1,000	1,026	1,239	993
1129 and 1139	Ca and P deficient in darkness	370	642	684	931	900	760	596	dead.

The food consumption of the pigs receiving a Ca and P low diet but allowed access to sunlight was not significantly different from that of the controls during the first two months of the experimental period but after that period the difference became increasingly greater. Nos. 1129 and 1139 showed poor consumption of food sooner and were significantly different from their group mates and also from the controls in this respect during the third month of the experiment.

The values for the inorganic P and the phosphatase content of the serum are given in Table XI.

The inorganic P content of the serum of Nos. 1132 and 1129 was lower than that of the controls but the reduction was not as marked as when P alone was deficient (Experiment I). The phosphatase values also indicated poorer calcification when P and Ca were deficient than in the control pigs. The blood Ca values gave an average of 9·5 in February, 1936, which was not significantly different from 9·7 obtained for the controls.

The results of the analysis of the femur are given in Table XII.

The percentage ash calculated on the dry fat free bone of the pigs receiving low P and Ca diets was reduced and considerably so in the case of No. 1129 which remained in semi-darkness during the experiment. The values suggest less bone material when Ca and P were present in insufficient amounts even if the ratio of Ca : P in the diet was a favourable one.

Summarizing the data presented it would appear that the presence of light was responsible for the differences in body weight and food consumption between the two pairs of pigs receiving rations low in both Ca and P, but it should be remembered that a

TABLE XI.

Phosphatase (Bodansky Units) and Inorganic Phosphorus (I.P.) (mgms. per 100 ml. Serum).

No. of Pig.	Experimental detail.	7 11 35.		4 12 36.		21 1 36.		11 2 36.		23/3, 36.		5/5/36.	
		Ph.	I.P.	Ph.	I.P.	Ph.	I.P.	Ph.	I.P.	Ph.	I.P.	Ph.	I.P.
1138	Control pig in darkness	7·2	8·7	5·3	8·0	7·6	9·0	7·0	8·1	9·4	8·0	10·0	8·2
1133	Control pig in light	7·4	8·0	10·7	7·8	12·2	9·2	—	7·8	11·8	8·6	12·1	9·1
1132	Ca and P low (in light)	8·0	—	10·7	—	18·7	6·3	—	4·1	—	—	22·4	5·6
1129	Ca and P low (in darkness)	9·8	7·2	14·1	6·8	15·7	5·5	—	4·1	19·5	6·0	15·8	—

TABLE XII.

Analysis of femur.

No. of pig.	Experimental details.	Green weight. Gms.	On green weight.		On dry fat free bone.			On ash.		
			% fat and water.	% dry fat free bone.	% ash.	% Ca.	% P.	% Ca.	% P.	Ca : P.
1133	Control in light	100·1	57·6	42·4	62·3	23·7	11·6	38·1	18·6	2·06 : 1
1140	Control in darkness.	106·2	58·2	41·8	61·4	22·9	11·3	37·3	18·4	2·03 : 1
1132	Ca and P low in light	109·0	76·1	23·9	57·9	20·9	10·5	36·1	18·1	2·00 : 1
1129	Ca and P low in darkness	77·4	70·9	29·1	51·7	18·4	9·6	35·6	18·6	1·91 : 1
1139	Ca and P low in darkness	149·4	72·2	27·8	50·9	19·0	9·3	37·3	18·2	2·05 : 1

decided difference between the two groups, as far as body weight was concerned began to show only after one pig had been killed in each group during February, leaving only one pig for comparison. For this reason no conclusions can be drawn and further work has been undertaken to elucidate the point.

It should be pointed out that when either Ca or P was deficient no difference appeared to exist when light was excluded in spite of the abnormal ratio, but growth was considerably slower in these cases than in the pigs receiving a ration deficient in both Ca and P. More rapid growth in the latter case might have been responsible for the differences observed when light was excluded and for the difference in the clinical picture presented by the pigs, but surmise is dangerous at this stage.

No. 1132, allowed access to light and killed in February for bone studies, appeared clinically healthy throughout, with perhaps only a suggestion of being slightly stiff. Its group mate, No. 1141, which remained in the experiment until it was discharged in June, first showed signs of stiffness in March and remained so until the end.

In contrast with the above, the pair on the same ration but kept in semi-darkness fared less well. No. 1128 appeared weak on the legs at the end of January and lay down most of the time when not feeding. Especially the hind legs seemed to be unable to carry the weight of the body. During February it was very stiff and remained down when feeding. It could rise, however, when urged to do so. This pig was killed towards the end of February for bone studies. Its group mate, No. 1139 developed stiffness during February and became very stiff in March. Towards the end of March it had to struggle fiercely when rising. During April it was hardly able to walk after a struggling attempt to rise and stood shivering most of the time. Its condition became worse and the animal showed nervous symptoms in addition. It would scream continuously when approached and seemed helpless to move except with the greatest effort. Its legs were so stiff that it could not take longer steps than a couple of inches at a time. This pig died on 27.5.36 and the bones were collected for analysis. Histological sections of the 3rd rib of No. 1139 were examined microscopically both in March, when a portion of the costochondral junction was removed, and after death in May. In March marked bone atrophy was noticed but although some red osteoid seams were present the amount did not transgress physiological limits and a diagnosis of rickets was not justified. In May the bones still showed marked bone atrophy but no rickets and it would be difficult to compare the difference in degree of bone atrophy between the two pigs without a more detailed microscopical study of all the bones. It would seem, however, that the extra three months in the experiment provided insufficient time for the development of rickets or osteofibrosis under the conditions of Ca and P intake that existed in the experiment.

Nos. 1132 and 1141, kept under the same conditions of Ca and P deficiency as 1139 and 1192 but allowed free access to light, appeared to show better growth, as already stated. The rib sections of No. 1132, killed in February, showed marked bone atrophy and the presence of some red seams of osteoid but hardly rickets. Rib sections cut in June from the experimental mate of No. 1132, viz. 1141, also showed marked atrophy and more osteoid than normal or incipient rickets.

The clinical picture presented by the two pairs of pigs on Ca and P low diets favours the conclusion that the absence of light affected the one pair of pigs detrimentally. The same indication is found by examining the body weights, food consumption and some of the other data. The histological findings bear out the same point, viz. that the pair in the light developed indications of rickets which might follow better growth, while the pair in the dark showed no rickets, but as already stated the evidence is too limited to justify conclusions.

A fact which stands out clearly, however, and which must be emphasised is that Ca deficiency with excess P, and P deficiency with Ca excess, affected the pigs of both the ages used in these experiments more detrimentally as far as all the observations made were concerned than when the ration was deficient in both Ca and P. The abnormal ratio of Ca to P when only one of these constituents was present in insufficient amounts was undoubtedly responsible for the more drastic effects registered. It would appear, however, that rickets which develops rapidly when the P content of the ration is low is not produced as easily in pigs when both Ca and P are low. This observation does not apply to sheep, goats and bovines in which rickets is easily produced when both Ca and P are low in the diet. The possibility remains, however, that reduced quantities of P and Ca will produce rickets in pigs, as it should, in virtue of the low P, even when both are present in deficient amounts and in a favourable ratio.

Furthermore, as the ratio of Ca to P in the feed of pigs kept on rations deficient in one or both of the two constituents was mainly responsible for the differences registered between pigs receiving a normal and abnormal ratio respectively, it was decided to determine the effect of an abnormal ratio of Ca to P when these elements were present in sufficient amounts.

EXPERIMENT IV.

The Ration contained Excess of either Ca or P and a Sufficiency of the other Mineral; in Other Respects it was Adequate.

Two pairs of six-months-old pigs were selected and given the same basal ration as that reported in Experiment I for pigs of this age, viz.:—

100 ml. milk,

100 gms. green feed.

Mash according to appetite and consisting of 90 parts maize samp, 10 parts high protein meatmeal and 1 part of salt.

Calcium carbonate and disodium phosphate were added to the milk of the pigs but in such a manner that the one pair, Nos. 995 and 984, received sufficient Ca and excess P. viz. 6 gms. and 8·2 gms. respectively, while the other pair, Nos. 977 and 997, received sufficient P and excess Ca, viz. 3 gms. and 13·7 respectively. The ratios of Ca to P in the feed of the two pairs were 1:1·4 and 1:0·22.

The experiment began in January, 1934, and was concluded approximately nine months afterwards. All the pigs consumed their food at least as well as the controls did and at no stage did any show symptoms of abnormalities.

The curves representing the average body weights of the two pairs of pigs compared with the controls are given in Figure 11.

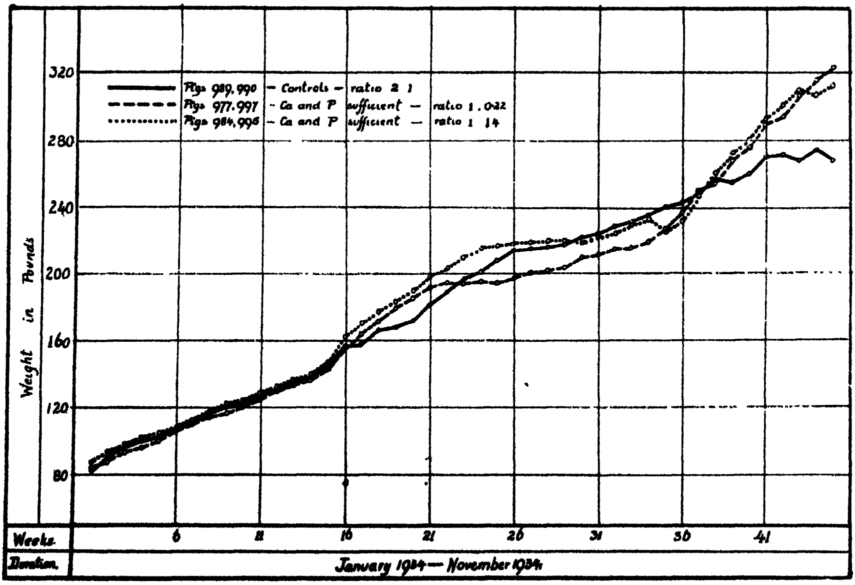

Fig. 11.

The excess of Ca in the feed of the one pair of pigs and the excess P in that of the other were apparently without detrimental effect on the weight increase of the pigs. It was only during the last eight weeks of the experiment, after one pig of each pair had been killed for bone studies, that the remaining experimental pigs appeared to increase more rapidly in weight than the control pig; this result cannot be regarded as significant therefore.

The inorganic P content of the blood determined four months after the beginning of the experiment show no difference between the control group and the one receiving enough P and excess lime. The values are 7·1 and 7·5 mg. P per 100 ml. blood for these two groups respectively. The inorganic P content of the blood of the pair receiving excess P and sufficient lime was 8·9 mgm. p.c. Blood calcium was normal in all three pairs, viz. 10·2 to 10·8 mgm. p.c.

The results of the analyses of the femurs are given in Table XIII.

TABLE XIII.

On Green Weight.

Nos.	Experimental details.	Green weight.	% fat and water.	% dry fat free bone.	Specific gravity.	% ash in dry fat free bone.
989	Control............	278·3	52·2	47·8	1·13	62·5
990	Control............	380·0	51·6	48·8	1·07	63·1
995	Suff. Ca excess P...	359·0	49·1	50·9	1·12	64·2
977	Suff. P excess Ca...	370·0	53·0	47·0	1·07	63·4
997	Suff. P excess Ca...	287·8	50·6	49·4	1·05	62·3

It is obvious from the figures given in Table XIII that not one of the values given was affected significantly by excess lime or P in the ration. The femurs of the pigs receiving excess Ca or P did not contain less bone material than those of the controls and as far as could be ascertained the bones were normal.

The histological sections of the ribs examined microscopically revealed no abnormalities.

It would appear therefore that if a sufficieney of P or Ca be given in the diet of six-months-old pigs an excess or Ca or P to the extent of that given in this experiment, viz. almost one and a half times as much P as Ca and in another case three and a half times as much Ca as P is without significant effect on body weight, food consumption, blood and bone analyses. In contrast to the above an abnormal ratio of Ca to P under conditions of Ca or P deficiency had disastrous effects on body weight, food consumption, blood and bone analysis of pigs as against less detrimental effects when the ratio was corrected but the deficiency retained as reported in Experiments I, II, III.

Although the experiment reported here by no means exhausts the study of the effect of abnormal ratios of Ca:P under conditions of a sufficiency of these two minerals it does suggest that an abnormal ratio under these conditions may not be as important as under conditions of a deficiency of P or Ca and work along these lines is being undertaken at present. Furthermore, arising out of the results of this work, it was decided to test the effect of an abnormal Erdalkali-Alkalizität in the ration of pigs under conditions of a sufficiency of Ca and P. Obviously changes in E.A. ($CaO+MgO-P_2O_5$ in mgm. equivalents per 100 gms. dry feed) involve changes in the Ca:P ratio.

EXPERIMENT V.

The rations contain a sufficiency of Ca and P but are abnormal with respect to the Erdalkali-Alkalizität in Marek's sense.

Briefly, Marek and his co-workers (1932 and 1935) claim that the best response to an adequate ration is obtained when the Erdalkali-Alkalizität lies between 20 and 25 mgm. equivalents. Furthermore, that if the E.A. is far removed from what they regard as the normal limits (20-25 mgm.) osteodystrophic disease is practically certain to develop. From a careful consideration of Marek's work it would appear that his results were obtained under conditions of vitamin D deficiency. If this is the case his results are not directly comparable with most of those reported in this experiment where vitamin D was present in abundance, nor is it then correct to claim that an abnormal E.A. is the sole cause of osteodystrophic disease is his experiments as, obviously, vitamin D deficiency was a complicating factor.

It should be pointed out that many rations which are considered normal are not such in Marek's sense and that unless special attention is paid to the E.A. of a ration the Erdalkali-Alkalizität most probably does not lie between the limits accepted by Marek. The control group in this experiment for instance showed normal growth and remained normal throughout the course of the experiment but did not receive a ration of which the E.A. lay between 20 and 25 mgm. equivalents. This point will be brought out more clearly, however, when considering the various rations fed.

Another factor which was considered important in this experiment was the production of rickets. Up to the present rickets has been produced here in pigs receiving adequate vitamin D only when the ration was deficient in phosphorus. This again is apparently at variance with Marek's work in which rickets was reported in pigs receiving sufficient P in their diet stated to be adequate in all respects except its E.A. Hence two of the rations given in our experiment corresponded as closely as practicable with the diets used by Marek and on which he produced severe rickets, our object was to produce rickets which could be proved to be due to a cause other than P deficiency.

The general treatment of the pigs was the same as that reported in Experiment I, and the experiment began in April, 1935, with 11-weeks-old pigs and lasted until the end of August.

The control pigs, Nos. 1076 and 1079, were given 100 ml. milk, 100 gms. green feed and mash according to appetite. The mash consisted of 94 parts maize samp, 6 parts bloodmeal, and 1·5 parts of salt. $CaCO_3$ and Na_2HPO_4 were added to the mash to ensure a daily intake of 7·2 gms. CaO and 6·3 gms. P_2O_5; the daily food supplied contained on an average ·44 gms. MgO. The Erdalkali-Alkalizität for the entire period was +6·5 mgm. equivalents.

These pigs weighed 13·3 Kg. on an average at the beginning of the experiment ond 68·4 Kgs. four months afterwards when they were discharged. Their appetite was good throughout and no indication of disease was noticed at any stage.

Radiographs were taken approximately two months after the beginning of the experiment and No. 1079 was killed an 30.8.35, for bone studies, i.e. after having been 120 days in the experiment.

The bones showed normal growth and development histologically and appeared to be perfectly normal from the radiographs. The percentage ash registered in the dry fat free bone was 54·8.

Several other pairs of pigs of the same age were fed concurrently with the pair mentioned above on rations showing a variation in their Erdalkali-Alkalizität as indicated below.

Nos. 1085 and 1090 were given a mash according to appetite and consisting of:—

Yellow maize meal	11 lb.
Barley meal	55 ,,
Wheaten bran	11 ,,
High protein meatmeal	11 ,,
Salt	1·5 ,,

$CaCO_3$ and Na_2HPO_4 were added to the daily ration to ensure a daily intake of CaO and P_2O_5 per 10 Kg. body weight of 10·8 and 4 gms. respectively. The ratio of CaO to P_2O_5 was 1:2·7 mgm. The Erdalkali-Alkalizität varied from 62 to 7·6 mgm. equivalents; the protein intake was 55 gms. per 10 Kg. live weight.

Another pair of pigs of the same age, No. 1084 and 1086, were given the same ration as that mentioned above, but these pigs were kept indoors permanently. It should be mentioned, however, that no attempt was made to exclude any but direct light and that it cannot be stated that this pair of pigs was kept under conditions of vitamin D deficiency. In addition the feed given, being grown under South African conditions and therefore exposed almost daily for 5 to 10 hours to direct sunlight during the growing and ripening period probably also supplied appreciable quantities of vitamin D. Still, direct sunlight was excluded to reduce the vitamin D present.

Another pair of pigs, viz. Nos. 1082 and 1087, was given the same mash as the two last mentioned pairs but 100 gms. green feed and 500 c.c. milk were given daily in addition to the quota of mash to improve the quality of the feed. $CaCO_3$ and Na_2HPO_4 were again added and the average E.A. for the entire period was found to be 65 mgm. equivalents.

Yet a fourth pair of pigs, viz. Nos. 1088 and 1074, was given the same mash which, incidentally, was responsible for the production of very severe rickets by Marek. Less $CaCO_3$ was added to the mash of this pair of pigs in order to create a more favourable Ca:P ratio and $Mg(OH)_2$ was added to the daily ration to keep its E.A. approximately the same as that of the other groups receiving this mash. The average E.A. for the period for this pair of pigs was 61 mgm. equivalents, and the CaO:P_2O_5 ratio 1·3 to 1 with an average intake of 11·5 gm. CaO and 9·0 gm. P_2O_5.

Results.

The weights of the individual pairs of pigs and the averages for each pair are given in Table XIV submitted below.

TABLE XIV.

Weights, given in Kgms.

1935.

Nos.	E.A.	12/4	15/4	25/4	29/4	7/5	13/5	20/5	27/5	3/6	10/6	17/6	24/6	1/7	8/7	15/7	22/7	30/7	6/8	13/8	19/8	26/8
1076......	+6·5	14·9	14·0	15·6	16·6	17·7	19·5	20·6	23·1	24·9	29·2	32·6	37·1	42·5	46·9	46·4	55·6	64·1	68·1	71·9	75·6	79·2
1079......	+6·5	11·8	11·3	12·2	13·1	13·1	12·7	14·0	15·4	16·8	17·7	19·1	22·9	27·6	30·5	35·4	39·3	43·1	47·2	49·6	53·6	57·6
Average.....		13·35	12·6	13·9	14·8	15·4	16·1	17·3	19·25	20·8	23·45	25·8	30·0	35·05	38·7	40·9	47·45	53·6	57·6	60·7	64·6	68·4
1085......	+67	7·3	7·3	7·9	8·4	9·5	10·9	12·4	14·5	17·3	20·0	23·6	26·9	30·1	31·9	35·3	36·2	43·6	46·0	50·2	52·9	52·9
1090......	+67	6·6	6·4	7·3	7·7	8·8	9·5	11·1	13·1	15·6	17·7	20·4	23·3	26·3	27·3	30·1	32·3	37·7	38·9	41·3	42·9	46·3
Average.....		6·9	6·8	7·6	8·05	9·15	10·2	11·7	13·8	16·45	18·8	22·0	25·1	28·2	29·6	32·7	34·2	40·6	42·45	45·7	47·9	49·6
1086......	+67	7·0	7·3	7·7	8·2	9·5	10·0	11·8	13·3	15·9	19·1	22·2	24·0	27·2	30·8	32·6	34·6	38·9	40·4	43·6	44·7	47·4
1084......	+67	7·7	7·9	8·8	9·1	10·4	11·3	12·9	14·5	17·5	20·0	23·3	24·9	28·1	31·2	32·8	35·7	40·2	42·9	45·8	48·1	55·4
Average.....		7·35	7·6	8·25	8·6	9·9	10·6	12·35	13·9	16·7	19·5	22·7	24·45	27·6	31·0	32·7	35·1	39·5	41·6	44·7	46·4	51·4
1082......	+65	8·2	8·2	9·1	9·5	10·9	12·0	13·1	14·2	17·9	21·8	25·4	29·2	32·3	37·1	43·1	49·2	55·6	57·2	59·4	61·0	64·9
1087......	+65	7·5	7·7	8·6	9·1	10·4	11·3	13·1	14·2	17·0	19·1	23·1	26·3	29·4	33·0	36·8	39·8	45·6	46·7	49·6	52·2	56·9
Average.....		7·8	7·9	8·8	9·3	10·6	11·6	13·1	14·2	17·45	20·45	24·25	27·7	30·8	35·0	39·9	44·5	50·6	51·9	54·5	56·6	60·9
1088......	+61	8·2	7·9	8·8	9·3	10·9	12·0	14·2	15·1	18·2	22·2	25·6	29·0	33·7	36·2	43·5	46·5	53·3	55·6	60·5	62·8	65·6
1074......	+61	10·6	10·2	10·9	11·3	13·1	14·0	14·9	17·9	20·9	25·1	28·3	33·0	37·5	41·3	47·2	51·8	59·4	60·1	65·6	66·1	71·0
Average.....		9·4	9·0	9·8	10·3	12·0	13·0	15·0	16·5	19·5	23·6	26·9	31·0	35·6	38·7	45·3	49·1	56·8	57·8	63·0	64·45	68·3

A statistical analysis of the results shows that no significant differences existed between the group weights at the end of the experimental period.

None of the groups showed an adverse reaction to the conditions of the experiment. The food consumption records show no significant differences between the groups and all the pigs remained healthy throughout the course of the experiment.

X-ray photographs were taken one month before the conclusion of the experiment and revealed no differences in bone structure in the comparative groups.

One pig of each pair was killed at the conclusion of the experiment, i.e. after 122 days experimental period, and rib sections were examined histologically. These sections were found to be normal and revealed normal bone growth and development in every case.

DISCUSSION.

There is no doubt that under the conditions described, rickets was not produced in pigs which received for 122 days rations so compounded that their Erdalkali-Alkalizität was abnormal in Marek's sense. Furthermore, there was apparently no difference in the body weights, food consumption and microscopical bone structure between the pigs receiving a ration whose E.A. was +6·5 mgm. equivalents when compared with those on a ration of approximately 65 mgm. equivalents.

As vitamin D was present in abundance in the experiment reported here, whereas the same ration used by Marek contained negligible quantities of this vitamin the conclusion appears justified that the difference between our results and those of Marek is due to the difference in the Vitamin D content of the ration used by the two schools of workers. It is also obvious that the highly positive E.A. in Marek's experiment wos not responsible per se for the production of osteodystrophic disease. It would appear unwise, therefore, to emphasize the necessity of a correct Erdalkali-Alkalizität for normal growth and development in pigs unless it is stated that the E.A. of a ration is important when pigs are kept under conditions of vitamin D deficiency.

Concurrently with the above two pairs of pigs of the same age and litters as those used were given another of Marek's rations whose Erdalkali-Alkalizität was -25 mgm. equivalents and which produced severe rickets after 141 days.

The ration consisted of 500 ml. separated milk, crushed maize ad lib to which bonemeal and salt were added to ensure a daily intake per 10 Kg. body weight of approximately 35 gms. protein, 5·5 gms. CaO, 7 gms. P_2O_5 and an Erdalkali-Alkalizität of -25 mgm. equivalents.

This ration, as stated by Marek, is low in protein which was poor in quality. The pigs did not respond well and scouring was frequently present for short periods. Growth was poor on the whole and the pigs lagged behind those already mentioned and receiving the ration whose Erdalkali- Alkalizität was highly positive. The fortnightly body weights of these pigs compared with those of the controls are given below:—

TABLE XV.

1935.

Nos.	E.A.	12/4	24/5	7/5	20/5	3/6	17/6	1/7	15/7	30/7	13/8	26/8
1076..........	+6·5	14·9	15·6	17·7	20·6	24·9	32·6	42·5	46·4	61·1	71·9	79·2
1079..........	+6·5	11·8	12·2	13·1	14·0	16·8	19·1	27·6	35·4	43·1	49·6	57·6
Average....		13·35	13·9	15·4	17·3	20·8	25·8	35·0	40·9	53·6	60·7	68·4
1078..........	—25	11·1	12·2	14·2	16·4	18·2	23·1	26·7	30·8	34·6	36·8	43·8
1080..........	—25	9·3	10·2	12·9	13·6	16·1	18·2	21·3	23·6	26·7	29·4	30·8
1081..........	—25	9·7	11·3	13·3	15·1	17·0	19·5	23·6	27·6	29·6	33·7	36·3
1083..........	—25	8·6	10·2	12·0	12·4	12·9	15·4	17·9	20·5	22·9	24·9	27·6
Average....		9·9	10·9	13·1	14·3	16·05	19·05	22·4	25·6	28·4	31·2	34·4

The body weights of the pigs receiving the maize ration reveal, as would be anticipated, poor weight increase, viz. 24·5 lb. after four months, compared with 55 lb. for the controls. The pigs showed poor appetites from time to time and although obviously hungry did not relish their food. The radiographs taken after three months in the experiment show bone atrophy and the rib sections of pigs Nos. 1080 and 1083 killed after 122 days in the experiment confirm this finding. Osteoporosis was not extensive, but certainly present, while the amount of osteoid present did not transgress physiological limits; osteofibrosis and rachitic lesions were absent . It is not surprising that bone formation did not proceed normally if the inadequacy of the ration both in regard to the quality and the quantity of the protein is considered. The point that is most important, however, is that in spite of a strongly negative Erdalkali-Alkalizität neither rickets nor osteodystrophia fibrosa was produced as was found to be the case by Marek, working with the same diet but probably under conditions of vitamin D deficiency.

For the convenience of the reader the main features of the five experiments reported in this publication are shown below in tabular form. (Table XVI.)

A glance at the table given above reveals the fact that osteodystrophic diseases were produced only when the intake of P or Ca or both was low and that bone disease was absent whenever sufficient quantities of these minerals were supplied in spite of the Erdalkali-Alkalizität of the ration ranging from - 9 to + 65 milligram equivalents.

Here again, as in the case of a ration of strongly positive E.A., it would appear that the difference in the results obtained at this Institute when compared with those of Marek and his school lies in the difference between the vitamin D contents of the respective rations.

TABLE XVI.

No. of experiment.	Special feature of ration.	No. of Pigs.	Age at beginning of experiment.	Daily intake (Percentages.)		Ratio. Ca : P.	Vitamin D. content.	Erdalkalialkalizität. (Mgm. equivalents.)	Diagnosis.
				Ca.	P.				
I	P low	2	6 mths.	0·67	0·09	7·5 : 1	Sufficient	+30	Rickets.
I	Normal	2	6 mths.	0·4	0·3	2 : 1	"	+ 5	Normal.
I	P low	2	8 wks.	2·0	0·11	18·4 : 1	"	+92	Rickets.
I	Normal	2	8 wks.	1·0	0·55	1·8 : 1	"	+2·3	Normal.
I	P low	2	8 wks.	2·0	0·11	18·4 : 1	In semi-darkness	+92	Rickets.
I	Normal	2	8 wks.	1·0	0·55	1·8 : 1	"	+2·3	Normal.
II	Ca low	2	6 mths.	0·06	0·6	1 : 10	Sufficient	−60	Osteoporosis, no rickets.
II	"	2	8 wks.	0·11	0·98	1 : 9	"	−86	" "
II	"	2	8 wks.	0·11	0·98	1 : 9	In semi-darkness	−86	" "
II	"	2	8 wks.	0·04	0·6	1 : 15	Sufficient	−50	(*a*) One died early in experiment. (*b*) Osteofibrosis in remaining pig.
III	Ca & P low	2	6 mths.	0·06	0·04	1·5 : 1	Sufficient	+ 2	Osteoporosis and slight rickets.
III	"	2	8 wks.	0·11	0·11	1 : 1	"	− 2	Osteoporosis and slight rickets.
III	"	2	8 wks.	0·11	0·11	1 : 1	In semi-darkness	− 2	Osteoporosis but no rickets.
IV	Sufficient Ca & P in abnormal ratios	2	6 mths.	0·40	0·8	1 : 2	Sufficient	−19	Normal.
IV		2	6 mths.	1·1 CaO grms. (per 10 kg.	0·2 P_2O_5 body wt.)	5·5 : 1 Ratio	"	+23	Normal.
V	Abnormal E.A.	2	11 wks.	7·2	6·3	1·1 : 1	Sufficient	+ 6·5	Normal.
V	"	2	"	10·8	4·0	2·7 : 1	"	+62	Normal.
V	"	2	"	"	"	1·7 : 1	Probably sufficient	+62	Normal.
V	"	2	"	"	"	2·7 : 1	Sufficient	+62	Normal.
V	"	2	"	11·5	9·0	1·3 : 1	"	+61	Normal.
V	"	4	"	5·5	7·0	1 : 1·3	"	−25	Slight osteoporosis.

The control pigs to those mentioned in Experiments II, III and IV are those of the relative ages given as "Normal" in Experiment I.

Rations of strongly positive or negative Erdalkali-Alkalizität apparently do not produce rickets or osteodystrophia fibrosa in young, growing pigs if sufficient P and Ca are present, even after several months' feeding, unless the vitamin D content of the ration is below the requirements of the pigs. Marek's results therefore cannot be described as being directly due to the Erdalkali-Alkalizität of the ration but were apparently due to a complex of at least two factors, viz. E.A. and vitamin D shortage.

SUMMARY AND CONCLUSIONS.

1. Data are presented on the effect upon growing pigs of rations deficient in P or Ca or both. Observations were made upon the body weight, food consumption, blood analysis and bone analysis—physical, chemical and histological.

2. The effects of the Ca : P ratio and of the Erdalkali-Alkalizität of some of the rations were also considered and discussed.

Low P, Abnormal Ratio.

3. ·8gm. of P and ·1 per cent. P were found to be not only insufficient for the normal growth and development of young growing pigs but caused severe rickets as determined microscopically.

4. The absence or presence of light was apparently without influence on the effects of the phosphorus deficiency and this suggests that the ration, probably already contained a sufficiency of vitamin D.

5. The low P content of the ration was reflected as low inorganic phosphorus in the serum, while serum phosphatase (Bodansky units) gave significantly higher than normal values, suggesting poor calcification.

6. A considerably smaller percentage of ash was present in the bones of the pigs suffering from P deficiency than in those of the control group.

7. Clinical symptoms of P deficiency appeared shortly after the beginning of the experiment and gradually became more pronounced until severe rickets could be diagnosed with the naked eye, when the pigs were in a pitiable state and the experiment discontinued. The addition of phosphate to the ration at this stage cured the rickets but the pigs remained smaller than the controls and had a stunted appearance, nor did the legs become normal.

Low Ca, Abnormal Ratio.

8. ·1 per cent, Ca in one experiment and 1 gm. daily in another were found to be insufficient for the normal growth of young pigs.

9. As in the case of P deficiency, light appeared to be without effect on the results of the Ca deficiency and it is surmised that the food being grown under conditions of abundant sunshine, contains sufficient vitamin D for the requirements of the pigs.

10. Neither serum phosphatase nor serum Ca was significantly affected by the low Ca of the ration.

11. The ash of the bones was considerably reduced.

12. Bone atrophy or osteoporosis was present but no rickets. In a third experiment in which the ration of a pair of pigs contained ·5 gms. CaO and 10·5 gm. P_2O_5 the bones of the surviving pig showed incipient osteodystrophia fibrosa and bone atrophy after 105 days but no indications of rickets. The suggestion is made that Ca deficiency per se will not produce rickets, but might produce osteodystrophia fibrosa in pigs and further work along these lines is being carried out.

Ca and P Deficiency, Normal Ratio.

13. 0·1 gm. Ca and ·6 gm. P were contained in the daily ration in one experiment and ·11 per cent. of Ca and P respectively in another experiment.

14. The weight increase of the pigs was unaffected for the first twenty weeks of the experiment when the animals on the Ca and P deficient ration began to increase more slowly in weight which differed significantly from that of control pigs at the end of the experiment after approximately another twenty weeks.

15. The detrimental effect of Ca and P deficiency was by no means as severe as that of a deficiency of either Ca or P together with an excess of P or calcium respectively; the abnormal ratio of Ca : P in the latter case is believed to be responsible for the enhanced effect of the deficiency.

16. Food consumption was poorer than in the control group. percentage ash in the bones low and the inorganic P of the serum not as low as that of the pigs on P deficiency but Ca excess.

17. It would seem that the absence of light affected the health of the pigs detrimentally. The pig in the light showed at its worst only slight stiffness while that in semi-darkness could hardly stand and walked only with the greatest difficulty; its legs were very stiff and almost out of control.

18. Microscopically the ribs of all four pigs showed marked bone atrophy and a suggestion of rickets in two of the four animals. The suggestion is made that the deficiency of P and Ca was not acute enough or that the experiment was not conducted for a sufficiently long period to produce rickets or ostitis fibrosa or both. Work along these lines is being continued.

Ca and P Sufficiency with Abnormal Ratio.

19. A ration containing 6 gms. Ca and 8·2 gms. P was given to one pair of pigs while in the case of a second pair of animals the ration contained 13·7 gms. Ca and 3·0 gms. P.

20. Body weight, food consumption, blood analysis and bone composition, chemical and histological did not alter significantly when Ca and P were present in the ration in the proportions given above instead of that contained in the ration of the control group.

Erdalkali-Alkalizität (E.A.).

21. Rations whose average E.A. for the 120 days experimental period were 6·5 mgm. equivalents and approximately 62 mgm. equivalents respectively were fed to eleven weeks old pigs. Sufficient Ca and P were present for normal growth.

22. All the pigs grew normally and remained healthy, nor was there any significant difference in body weight, food consumption and bone development as judged radiographically and histologically.

23. Neither rickets nor osteodystrophia fibrosa developed as was reported by Marek on this ration.

24. It is suggested that the explanation of the apparently anomalous results lies in the fact that abundant vitamin D was present in our experiments while this was a limiting factor in Marek's experiments.

25. A strongly negative E.A. (– 25 mgm. equivalents) in a ration consisting of maize produced bone atrophy and no rickets. As maize protein is admittedly deficient in protein—qualitatively and quantitatively—the results obtained cannot be ascribed exclusively to the abnormal E.A. of the ration.

Marek produced rickets and osteofibrosis with this ration and the difference between his results and those obtained here is again ascribed to the difference in the Vitamin D content of the ration made up at the two Institutes.

26. It is suggested that the Erdalkali-Alkalizität of a ration when sufficient P and Ca are present is not exclusively responsible for the development or not of osteodystrophic diseases in pigs.

The writers wish to acknowledge gratefully the collaboration of Dr. Thomas, Mr. van der Wath, B.V.Sc., and Mr. B. A. du Toit, M.Sc. during the course of the investigation.

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The Digestibility of South African Feeds.

I. The Digestibility Coefficients of some Natural Grasses.

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VERY little information on the digestibility of South African grasses is available. As a matter of fact, work on the digestibility of South African feeds is practically non-existent and has long been overdue. Ross, Bosman and others, 1927 and 1931, determined the digestibility of lucerne hay, teff hay, and of maize oil cake and teff hay combined. The work was carried out with cattle and sheep as the experimental animals.

Sheep were used for the work reported in this communication mainly on account of the limited quantities of grass hay available. No repetitions were possible and the results should be regarded as suggestive rather than final.

The hays were obtained from the Rietondale Pasture Research Station and the work was carried out in co-operation with the Division of Plant Industry. Only four of the twelve grasses established on the acre plots yielded enough hay for the digestibility trials due to unfavourable climatic conditions. These were *Digitaria—Pretoria Large*, *Digitaria pentzii*, *Panicum makarikari* strain, and *Panicum phragmatoides*. These grasses were cut at the haymaking stage, dried and sent to Onderstepoort for the digestion trials.

Each acre plot was divided into four equal parts and each portion treated with fertilizer as indicated in the scheme submitted below:—

Type of Grass Hay.	Fertilizer Treatment.	Yield of Hay (℔.) per ¼ Acre.
I. (*a*) *Digitaria Pretoria Large*....	No fertilizer........................	172
(*b*) *Digitaria Pretoria Large*....	75 ℔. Superphosphate..............	385
(*c*) *Digitaria Pretoria Large*....	75 ℔. blood meal..................	222
(*d*) *Digitaria Pretoria Large*....	75 ℔. of a mixture of equal quantities of superphosphate and blood meal	746
II. (*a*) *Digitaria Pentzii*..........	No fertilizer........................	102
(*b*) *Digitaria Pentzii*..........	75 ℔. superphosphate..............	320
(*c*) *Digitaria Pentzii*..........	75 ℔. blood meal..................	392
(*d*) *Digitaria Pentzii*..........	75 ℔. of a mixture of equal quantities of superphosphate and blood meal	736
III. (*a*) *Panicum makarikari*......	No fertilizer........................	179
(*b*) *Panicum makarikari*......	75 ℔. superphosphate...............	507
(*c*) *Panicum makarikari*......	75 ℔. blood meal..................	280
(*d*) *Panicum makarikari*......	75 ℔. of a mixture of equal quantities of superphosphate and blood meal	610
IV. (*a*) *Panicum phragmatoides*....	No fertilizer........................	537
(*b*) *Panicum phragmatoides*....	75 ℔. superphosphate..............	738
(*c*) *Panicum phragmatoides*....	75 ℔. Ammonium sulphate....... ..	745

The fertilizers were applied after the grasses had been well established. The first application of fertilizer was made in September, 1932, which was repeated in November, 1933, and the grasses were cut for hay during the growing season of 1933-1934.

The rainfall for the individual months during 1932 and 1933, until February, 1934, is given below:—

	1932.	1933.	1934.
	Inches.	*Inches.*	*Inches.*
January..........................	2·79	1·12	7·5
February..........................	4·52	1·15	2·7
March..........................	3·56	2·76	—
April..........................	0·30	0·85	—
May..........................	0·55	0	—
June..........................	0	0·32	—
July..........................	0	0	—
August..........................	0	0	—
September..........................	0·43	0	—
October..........................	1·53	0	—
November..........................	0·83	13·1	—
December..........................	2·90	1·05	—

DETAILS OF THE DIGESTIBILITY TRIALS.

Ten mature wethers were selected for the trials. The sheep were placed in a small paddock where individual feeding boxes were available.

These ten sheep were healthy, feeding well and of fairly uniform body-weights. Prior to the first trial all sheep received lucerne hay *ad. lib.*

For the purpose of the collection of the faeces, each sheep was placed in metabolism harness.

All the weights of the sheep were taken, noted and tabulated, from time to time, as given in Table IV.

During the period of rest, the harness was discarded, and during the preliminary feeding period, the harness was replaced but the bags were left open; only during the actual experimental stage were the bags closed to ensure the collection of the faeces voided.

The First Trial.

The first trial began on the 8th October, 1934, the sheep on that day receiving 300 grams lucerne hay each; the next day the ration was increased to 400 grams, and, as the sheep consumed all with ease, it was again increased to 500 grams on the 15th October. Finally, the ration was raised to 600 grams daily per sheep and kept at that level; this level of feeding satisfied the maintenance need of each sheep.

The body-weights were taken on four occasions prior to the start of the preliminary period. The collection of the faeces was begun on the 26th October. The faeces were collected daily in the mornings at 8.30, and in the afternoons at 4; in this way, the bags were never too full or too heavy.

The animals were fed at 2 p.m. daily and each was kept in its individual crate overnight, and allowed to run free in the enclosure for exercise and water during the day, from about 8.30 to 2 p.m. This treatment was strictly adhered to both during the experimental and the preliminary-feeding periods.

All the weights of the individual collections of the faeces were taken and recorded.

From every two-day collections 10 per cent. samples were taken for chemical analysis; the water and the protein determinations were done immediately. The samples were air dried and stored in small jars for subsequent determinations.

Samples were taken of the lucerne hay fed during the trial and an average sample from the ten individual samples was analysed.

From the results of the analyses the coefficients of digestibility for the individual nutrients, and the nutritive ratio were calculated. From the body-weight figures,it was found that during this trial no loss in weight was recorded for the period 5th October to 7th November.

Sheep No. 38247 was withdrawn from the trial and no collection of its faeces taken; the faeces of sheep No. 38240 were left unanalysed. Hence, the results of 8 sheep were available for the first trial.

Between each trial a period of rest was allowed from 8th November to 19th November the sheep were rested and a daily ration of 600 grams lucerne hay for each sheep was continued.

The Second Trial.

On the 20th November the sheep were selected for the second trial, i.e. on grass hay of the species Digitaria-Pretoria Large (D.P.L.) and the animals were grouped as follows: Sheep Nos. 38245, 38249, 38239 were fed the hay (D.P.L.) from the unfertilized plot.

Sheep Nos. 38240, 38236, 38248 were fed the hay (D.P.L.) plot treated with superphosphate.

Sheep Nos. 38243, 38247, 38251 were fed the hay (D.P.L.) plot treated with blood meal.

The remaining hay of this Digitaria species plot, treated with the mixture of superphosphate and blood meal, was left over for the third trial.

The Third Trial.

The preliminary feeding period started on the 19th January, 1935, the sheep were placed on the grass hay and the harness adjusted. The period from the 19th to 28th was begun by feeding each sheep at the rate of 400 grams per day; on the 21st January the ration was 600 grams per day. The sheep were grouped as follows:—

Sheep Nos. 38245, 38249, 38239, 38252 were fed hay (*D. Pentzii*) from the unfertilized plot.

Sheep Nos. 38240, 38236, 38248 were fed the hay (*D. Pentzii*) from the plot fertilized with superphosphate.

Sheep Nos. 38243, 38247, 38251 were fed the hay (D.P.L.) from the plot fertilized with the mixture of superphosphate and blood-meal.

The experimental period started on the 28th January and the faeces collected twice daily until the 5th February, and as before, weighed, prepared for analysis and the weights recorded.

The body-weights of the sheep were taken, after the trial—on the 5th and the 8th—proving that the sheep had lost no weight throughout.

Unfortunately, sheep No. 38236 developed diarrhoea and had to be withdrawn from the experiment and again placed on lucerne hay *ad lib.*

The sheep were again rested from the experiment from the 5th February to 18 February and the harness removed. The sheep received the daily ration of 300 grams lucerne hay and 300 grams yellow maize per sheep. On the 15th February the ration was supplemented with a little green feed.

The Fourth Trial.

On the 18th February, the sheep were selected for this trial, and fed at the rate of 600 grams grass hay per sheep daily.

Sheep Nos. 38245, 38249, 38239, 38240 were fed the hay (*D. Pentzii*) from plot treated with blood meal.

Sheep Nos. 38236, 38248, 38243 were fed the hay *Panicum makarikari* (P. Mak.) from the plot unfertilized.

Sheep Nos. 38247, 38251, 38252 were fed the hay (*D. Pentzii*) from the plot fertilized with the mixture of superphosphate and blood meal.

All the sheep were again placed in harness. The body-weights were taken on consecutive days (20th, 21st and 22nd). The experimental period started on the 25th February and terminated on the 7th March. The faeces were collected twice daily, weighed and prepared for analysis.

The sheep did not lose in weight. Even the sheep on the fibrous, tough *Pan. makarikari* hay thrived, proving this hay not only to be of a good nutritional value, but also palatable.

Another rest-period followed from 8th February to 28th March; the harness was left off. The daily ration allowed was again 300 grams lucerne hay and 300 grams yellow mealies, per sheep. In addition a little green feed was given from the 9th to 24th March. The sheep were healthy throughout.

The ration was changed to 600 grams lucerne hay per sheep daily on the 25th March.

The harness was again replaced on the sheep, weighed on the 25th and 26th March.

The Fifth Trial.

On the 28th March the preliminary feeding was begun at the rate of 600 grams per sheep daily of the hays:—

Sheep Nos. 38245, 38249, 38239 were fed the hay *Panicum makarikari* (P. Mak.) from the plot treated with blood meal.

Sheep Nos. 38240, 38236, 38248 were fed the hay (P. Mak.) from the plot fertilized with the mixture of superphosphate and blood meal.

The experimental period lasted from the 9th April to the 17th April, and the faeces were collected. The sheep showed no loss in weight. A rest period was allowed from the 18th April until 27th May; 600 grams lucerne hay per sheep was given daily and the harness was removed. On the 4th May, the ration was changed to 300 grams lucerne hay and 300 grams yellow mealies.

On the 27th the ration was changed back to 600 grams lucerne hay.

The body-weights were registered on the 27th and the 28th May.

The Sixth Trial.

The preliminary feeding of the hays was started at the rate of 600 grams per sheep daily and the harness adjusted on the 3rd June.

Sheep Nos. 38245, 38249 were fed the hay *Panicum phragmatoides* (P. Phrag.) from the unfertilized plot.

Sheep Nos. 38240, 38247 were fed the hay (P. Phrag.) from the plot fertilized with superphosphate.

Sheep Nos. 38239, 38236 were fed the hay (P. Phrag.) from the plot fertilized with ammonium sulphate.

Sheep Nos. 38248, 38243, 38251, 38252 were fed lucerne hay.

The trial with lucerne hay as ration was made for the purpose of comparison with the previous trial in October (with the same animals).

A minimum quantity of the *Panicum phragmatoides* hays was available and for this reason only two sheep were allowed in each group. The body-weights taken before and after the trial showed no loss in weight. In the group receiving the lucerne hay, the sheep gained on an average 2 lb. each.

Hereafter the sheep were rested and discharged from the experiment.

RESULTS.

A. *The Coefficients of Digestibility.*

The weights of the different nutrients consumed, digested and voided (as faeces) by the individual sheep in each digestion trial, together with the coefficients of digestibility obtained for four different kinds of grass hays and lucerne hay, are given in the tables appearing in the appendix.

Two sets of coefficients of digestibility were obtained for lucerne hay; one set (using 8 sheep) at the commencement of the trials and another set of coefficients (using 4 sheep) at the conclusion of the trials. These values show remarkable similarity in digestibility by the different sheep used in these trials.

The digestibility coefficients given in the tables are those for dry matter, crude protein, crude fibre, nitrogen free-extractives, ash, organic matter, and of ether-soluble extracts. In Trial 2 with Digitaria Pretoria Large, grass hays as feed, it would appear that a general improvement in the digestibility was established by fertilizing the plots, especially marked in the case where the mixture of superphosphate and blood-meal had been given.

No marked improvements in digestibility could be found in the case of *Digitaria Pentzii*, even by fertilizing the plots. *Panicum makarikari* hays did not show wide variations in the digestibility values. This coarse hay was consumed with ease by all the sheep, proving that it was highly palatable. The *Panicum phragmatoides* of Trial 6, with a slightly higher protein content than any of the other grass hays, did not show a significantly higher digestibility coefficient for protein.

In Trial 6, the digestible values for the crude protein of a lucerne hay ranged from 63·4 to 71·0, with an average of 66·6, which does not differ from the average obtained with a lucerne hay in Trial 1.

Similarly, for the crude fibre, an average value of 45·5 was obtained in Trial 6, compared with an average of 43·0 in Trial 1.

B. *The Nutritive Ratios.*

From the available figures, the nutritive ratios have been calculated, and tabulated in the appendix.

The average nutritive ratio for lucerne hay in Trial 1 was 1:4·7 and for Trial 6 the ratio was 1:4·2. The difference in ratios for lucerne hay may be accounted for by the slightly higher protein content of the hay used in Trial 6. In the case of the Digitaria Pretoria Large species, narrower nutritive ratios were obtained for the hays from the plots treated with fertilizer than for the hay from the unfertilized plot.

In the case of *Digitaria pentzii,* however, the nutritive ratios remained approximately constant and it would appear that the fertilizers did not improve the digestibility of this grass significantly.

In the case of the *Panicum makarikari* the fertilizing appeared to have improved the digestibility.

The nutritive ratio obtained for *Panicum phragmatoides* was apparently also not significantly affected by the fertilizers.

The sheep throughout the different trials remained remarkably constant in body-weight and even after the lapse of the period from 7th November, 1934, to 22nd June, 1935, the increase in body-weight of each sheep was on an average only 7·8 lb., which included the growth of wool for that period.

SUMMARY.

Coefficients of digestibility and nutritive ratios are given for some South African grasses, as well as for lucerne hay.

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TABLE I.

Composition of Hays fed in Trials expressed in grams per cent.

Kind of Hay.	Plot Treated.	Trial No.	Moisture.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extract.	Organic Matter.
Lucerne (*A*)	—	1	7·4	14·1	36·0	2·24	8·9	38·8	83·7
Lucerne (*B*)	—	6	6·8	14·6	38·2	2·24	8·7	36·3	84·5
Digitaria Pretoria Large	Control	2	6·9	9·2	35·1	2·05	7·3	46·3	85·8
Digitaria Pretoria Large	Superphosphate	2	6·6	8·9	36·3	1·91	7·2	44·7	86·2
Digitaria Pretoria Large	Bloodmeal	2	6·7	10·2	34·6	1·85	7·3	46·1	86·0
Digitaria Pretoria Large	Bloodmeal and Superphosphate	3	6·8	10·1	35·4	2·03	6·5	46·0	86·7
Woolly Finger (*Digitaria Pentzii*)	Control	3	6·6	10·1	32·9	1·96	7·8	47·2	85·6
Woolly Finger (*Digitaria Pentzii*)	Superphosphate	3	6·6	8·9	34·9	2·04	7·2	47·0	86·2
Woolly Finger (*Digitaria Pentzii*)	Bloodmeal	4	6·0	9·6	35·9	1·95	7·4	45·1	86·6
Woolly Finger (*Digitaria Pentzii*)	Bloodmeal and superphosphate	4	7·0	9·8	33·3	2·17	6·3	48·4	86·7
Panicum makarikari	Control	4	6·9	7·1	34·3	1·94	4·4	52·3	88·7
Panicum makarikari	Superphosphate	5	6·7	7·2	34·6	2·06	4·5	51·6	88·8
Panicum makarikari	Bloodmeal	5	6·4	7·6	35·2	2·06	4·7	50·4	88·9
Panicum makarikari	Bloodmeal and superphosphate	5	6·8	7·6	34·1	2·13	5·1	51·1	88·1
Panicum phragmatoides	Control	6	7·1	11·4	34·1	1·89	6·4	46·2	86·5
Panicum phragmatoides	Superphosphate	6	6·9	11·3	34·4	1·73	6·0	46·6	87·1
Panicum phragmatoides	Ammonium sulphate	6	6·9	12·8	33·8	1·70	6·4	45·3	86·7

TABLE II.

Digestibility of Hay—Lucerne Hay.

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL I.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
Sheep No. 38245 (1).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2543·7	274·7	1226·1	73·8	195·9	773·3	2347·8
Nutrients Digested...	3012·3	508·7	774·1	50·6	299·5	1382·4	2712·8
Coefficients of Digestibility.............	54·2	64·9	38·7	40·6	60·4	64·1	53·6
Sheep No. 38249 (2).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2396·1	256·3	1160·0	69·4	201·5	708·0	2194·6
Nutrients Digested...	3159·9	527·1	840·2	55·0	293·9	1447·7	2866·0
Coefficients of Digestibility.............	56·8	67·3	42·0	44·3	59·3	67·0	56·8
Sheep No. 38239 (3).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2368·8	268	1110·0	88·4	213	700	2155·8
Nutrients Digested...	3187·2	515·4	890·2	36·0	282·4	1455·7	2904·8
Coefficients of Digesti-	57·4	65·7	44·5	29·1	57·1	67·7	57·4
Sheep No. 38236 (5).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2424·6	285	1160	77·4	208	704	2216·6
Nutrients Digested...	3131·4	498·4	480·2	47·0	287·4	1451·7	2844·0
Coefficients of Digestibility.............	56·2	63·6	42·0	37·9	58·0	67·2	56·2
Sheep No. 38248 (6).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2261·9	242	1105	63·1	194·5	656	2037·4
Nutrients Digested...	3294·1	541·4	895·2	61·3	300·9	1499·7	2993·2
Coefficients of Digestibility.............	59·1	69·0	44·7	49·3	60·6	69·4	59·1
Sheep No. 38243 (7).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2390·4	267·5	1120	69·3	244	712	2146·4
Nutrients Digested...	3165·6	515·9	880·2	55·1	251·4	1443·7	2914·2
Coefficients of Digestibility.............	57·0	65·8	44·0	44·2	50·7	67·0	57·5
Sheep No. 38251 (9).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2337·7	266	1125	74·6	213	656	2124·7
Nutrients Digested...	3218·3	517·4	875·2	49·8	282·4	1499·7	2935·9
Coefficients of Digestibility.............	58·0	66·0	43·8	40·0	57·0	69·4	58·0
Sheep No. 38252 (10).							
Nutrients Consumed..	5556	783·4	2000·2	124·4	495·4	2155·7	5060·6
Excrement...........	2233·2	244	1105	62·5	187·5	634	2045·7
Nutrients Digested...	3322·8	539·4	895·2	61·9	307·9	1521·7	3014·9
Coefficients of Digestibility.............	59·6	68·8	44·7	49·8	62·2	70·6	59·7
Average for 8 Sheep..	57·4	66·4	43·0	41·9	58·1	67·8	57·4

TABLE II—*(continued)*.

Coefficients: Digestibility of Hay—Digitaria Pretoria Large (Control).

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL II (*a*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38245 (1).							
Nutrients Consumed..	5586	513·9	1960·7	114·5	407·8	2591·3	5178·2
Excrement...........	2429	284·0	649	89·8	289	1120	2140·7
Nutrients Digested...	3156·3	229·9	1311·7	24·7	118·8	1471·3	3037·5
Coefficients of Digestibility.............	56·6	44·7	66·9	21·6	29·1	56·7	58·6
SHEEP No. 38249 (2).							
Nutrients Consumed..	5586	513·9	1960·7	114·5	407·8	2591·3	5178·2
Excrement...........	2373·3	273	600	80·6	296	1120	2077·3
Nutrients Digested...	3213·7	240·9	1360·7	33·9	111·8	1471·3	3100·9
Coefficients of Digestibility.............	57·4	46·8	69·4	29·6	27·4	56·7	59·9
SHEEP No. 38239 (3).							
Nutrients Consumed..	5586	513·9	1960·7	114·5	407·8	2591·3	5178·2
Excrement...........	2212·7	255·5	584·1	84·1	292	986	1920·7
Nutrients Digested...	3373·3	258·4	1376·6	30·4	115·8	1605·3	3257·5
Coefficients of Digestibility.............	60·3	50·3	70·2	26·5	28·4	61·8	62·9
Average (for 3 Sheep)	58·1	47·3	68·8	26·0	28·3	58·4	60·4

Coefficients: Digestibility of Hay—Digitaria Pretoria Large (Superphosphate).

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL II (*b*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38240 (4).							
Nutrients Consumed..	5604	488·7	2034·2	107·0	403·5	2505	5200·5
Excrement...........	2314·6	236·1	614	74·1	256·9	1131·8	2057·7
Nutrients Digested...	3289·4	262·6	1420·2	32·9	146·6	1373·2	3142·8
Coefficients of Digestibility.............	58·7	52·8	70·0	30·8	36·3	54·7	60·4
SHEEP No. 38236 (5).							
Nutrients Consumed..	5604	498·7	2034·2	107·0	403·5	2505	5200·5
Excrement...........	2287·7	240	638	82·4	275	1055	2012·7
Nutrients Digested...	3316·3	258·7	1396·2	24·6	128·5	1450	3187·8
Coefficients of Digestibility.............	59·2	51·9	68·6	23·0	31·7	57·8	61·3
SHEEP No. 38249 (6).							
Nutrients Consumed..	5604	498·7	2034·2	107·0	403·5	2505	5200·5
Excrement...........	2195·5	226	602	76·8	252	1040	1943·5
Nutrients Digested...	3408·5	272·7	1432·2	30·2	151·5	1465	3257·0
Coefficients of Digestibility.............	60·7	54·7	70·2	28·2	37·5	58·5	62·6
Average (for 3 Sheep)	59·5	53·1	69·6	27·3	35·2	57·0	61·4

TABLE II—*(continued).*

Coefficients: Digestibility of Hay—Digitaria Pretoria Large (Bloodmeal).

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL II (c).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38243 (7).							
Nutrients Consumed..	5598	571·0	1940	103·5	409	2580	5189
Excrement...........	2254·3	252	650	67·6	254	1025	2000·3
Nutrients Digested...	3343·7	319·0	1290	35·9	155	1555	3188·7
Coefficients of Digestibility..............	59·6	56·0	66·4	34·7	37·9	60·3	61·4
SHEEP No. 38247 (8).							
Nutrients Consumed..	5598	571·0	1940	103·5	409	2580	5189
Excrement...........	2313·4	262	625	69·3	277	1100	2036·4
Nutrients Digested...	3284·6	309·0	1315	34·2	132	1480	3152·6
Coefficients of Digestibility..............	58·5	54·1	67·8	33·0	32·2	57·4	60·7
SHEEP No. 38252 (10).							
Nutrients Consumed..	5598	571·0	1940	103·5	409	2580	5189
Excrement...........	2250	277	590	67·0	266	1035	198·4
Nutrients Digested...	3348	294·0	1350	36·5	143	1545	320·5
Coefficients of Digestibility..............	59·8	51·6	69·6	35·3	35·0	60·0	61·7
Average (for 3 Sheep)	59·3	54·0	68·0	34·3	35·0	59·2	61·3

Coefficients: Digestibility of Hay—Digitaria Pretoria Large (Bloodmeal and Superphosphate.)

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL III (a).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38243 (7).							
Nutrients Consumed..	4473·6	451·8	1583·6	90·8	290·8	2057·8	4182·8
Excrement...........	1526	164·5	427	51·8	180·0	702	1346
Nutrients Digested...	2947·6	287·3	1156·6	39·0	110·8	1355·8	2846·8
Coefficients of Digestibility..............	66·0	63·5	72·9	43·1	38·1	65·8	67·8
SHEEP No. 38247 (8).							
Nutrients Consumed..	4473·6	451·8	1583·6	90·8	290·8	2057·8	4182·8
Excrement...........	1559·3	171·5	441	48·3	189	710	1370·3
Nutrients Digested...	3914·3	280·3	1142·6	42·5	101·8	1347·8	2812·5
Coefficients of Digestibility..............	65·0	62·0	72·2	46·8	35·0	65·5	67·2
Average (for 2 Sheep)	65·5	62·7	72·5	45·0	36·5	65·7	67·5

Table II—*(continued).*

Coefficients: Digestibility of Hay—Digitaria pentzii *(Control).*

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL III (*b*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
Sheep No. 38245 (1).							
Nutrients Consumed..	4483·2	453	1475	87·7	349	2115	4134·2
Excrement...........	1689·1	183	452	48·7	218·5	778	1470·6
Nutrients Digested...	2794·1	270	1023	39·0	130·5	1337	2663·6
Coefficients of Digestibility..............	62·4	59·6	69·3	44·5	37·4	63·2	64·4
Sheep No. 38249 (2).							
Nutrients Consumed..	4483·2	453	1475	87·7	349	2115	4134·2
Excrement...........	1734·9	192·5	484	50·2	225·5	782	1509 4
Nutrients Digested...	2748·3	260·5	991	37·5	133·5	1333	2624·8
Coefficients of Digestibility..............	61·2	57·5	67·2	42·8	32·5	63 0	63 5
Sheep No. 38239 (3).							
Nutrients Consumed..	4483·2	453	1475	87·7	349	2115	4134·2
Excrement...........	1830·1	205	498	51·2	240	836	1590·1
Nutrients Digested...	2653·2	248	977	36·5	109	1279	2544·1
Coefficients of Digestibility..............	59·2	54·7	66·2	41·8	31·2	60·5	61·5
Sheep No. 38252 (10).							
Nutrients Consumed..	4483·2	453	1475	87·7	349	2115	4134·2
Excrement...........	1691·6	192·5	426	59·0	240	772	1451·6
Nutrients Digested...	2791·6	260·5	1059	28·7	109	1343	2682·6
Coefficients of Digestibility..............	62·3	57·4	71·8	31·8	32·0	63 5	64·9
Average (4 sheep)....	61·3	57·3	68·6	40·2	33·3	62·5	63·6

Coefficients: Digestibility of Hay—Digitaria pentzii *(Superphosphate).*

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL III (c).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
Sheep No. 38240 (4).							
Nutrients Consumed..	4483·2	399	1565	90·3	322·5	2105	4160·7
Excrement...........	1939·7	178·5	554	52·4	211	932	1728·7
Nutrients Digested...	2543·5	220·5	1011	37·9	101·5	1173	2432·0
Coefficients of Digestibility..............	56·5	55·2	64·6	42·0	31·5	55·7	58·4
Sheep No. 38252 (10).							
Nutrients Consumed..	4483·2	399	1565	90·3	322·5	2105	4160·7
Excrement...........	1617·3	147·2	479	45·2	216·5	730	1400·8
Nutrients Digested...	2765·9	251·8	1086	45·1	106·0	1375	2759·9
Coefficients of Digestibility..............	61·6	63·0	69·3	49·9	32·8	65·2	66·3
Average (for 2 Sheep)	59·1	59·1	66·9	46·0	32·1	60·5	62·3

TABLE II—*(continued).*

Coefficients: Digestibility of Hay—Digitaria pentzii *(Bloodmeal).*

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL IV (*a*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38245 (1).							
Nutrients Consumed..	5640	541·4	2020	110	417	2540	5223
Excrement..........	2614·6	274·0	752	60·2	295	1230	2319·6
Nutrients Digested...	3025·4	267·4	1268	49·8	122	1310	2903·4
Coefficients of Digestibility.............	53·6	49·3	62·7	45·3	29·2	51·6	55·6
SHEEP No. 38249 (2).							
Nutrients Consumed..	5640	541·4	2020	110	416	2540	5223
Excrement...........	2576·2	265	738	61·9	293	1230	2283·2
Nutrients Digested...	3063·8	276·4	1282	48·1	124	1310	2939·8
Coefficients of Digestibility.............	55·3	51·0	63·4	43·7	29·5	51·6	56·3
SHEEP No. 38239 (3).							
Nutrients Consumed .	5640	541·4	2020	110	417	2540	5223
Excrement...........	2309·9	235·5	654	55·4	261	1105	2048·9
Nutrients Digested...	3330·1	305·9	1366	54·6	156	1435	3174·1
Coefficients of Digestibility.............	58·8	56·5	67·6	49·7	37·4	56·4	60·8
SHEEP No. 38240 (4).							
Nutrients Consumed..	5640	541·4	2020	110	417	2540	5223
Excrement...........	2390	234·0	688	59·8	280	1130	2110
Nutrients Digested...	3250	307·4	1332	50·2	137	1410	3113
Coefficients of Digestibility.............	57·6	56·8	66·0	45·7	32·8	55·6	59·6
Average (for 4 Sheep)	56·3	53·4	64·9	46·1	32·2	53·8	58·1

TABLE II—*(continued)*.

Coefficients: Digestibility of Hay—Digitaria pentzii *(Bloodmeal and Super)*.

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL IV (*b*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38247 (8).							
Nutrients Consumed..	5580	547	1850	121·0	352	2700	5228
Excrement...........	2089·7	225·5	577	54·3	230	1005	1859·7
Nutrients Digested...	3490·3	32·5	1273	66·7	122	1695	3368·3
Coefficients of Digestibility.............	62·5	58·7	68·8	55·2	34·7	62·8	64·4
SHEEP No. 38251 (9).							
Nutrients Consumed..	5580	547	1850	121·0	352	2700	5228
Excrement...........	2281·5	235	648	61·6	233	1105	2048
Nutrients Digested...	3298·5	312	1202	59·4	119	1595	3180
Coefficients of Digestibility.............	59·0	57·0	65·0	49·0	33·8	59·0	60·8
SHEEP No. 38252 (10.)							
Nutrients Consumed..	5580	547	1850	121·0	352	2700	5228
Excrement...........	2134·2	213·4	576	62·0	237	1045	1897·2
Nutrients Digested...	3445·8	333·6	1274	59·0	115	1655	3330·8
Coefficients of Digestibility................	61·7	61·0	68·8	48·8	32·7	61·2	63·7
Average (for 3 Sheep)	61·0	59·0	67·5	51·0	33·7	61·0	63·0

Coefficients: Digestibility of Hay—Panicum makarikari *(Control)*.

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL IV (*c*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38236 (5).							
Nutrients Consumed..	5586	396	1915	108·2	245	2920	5341
Excrement...........	2372	187·5	908	73·4	147	1055	2225
Nutrients Digested...	3214	208·5	1007	34·8	98	1865	3116
Coefficients of Digestibility.............	56·6	52·7	52·6	32·2	40·0	63·8	58·3
SHEEP No. 38248 (6).							
Nutrients Consumed..	5586	396	1915	108·2	245	2920	5341
Excrement...........	2263	181	836	65·5	145	1035	2118
Nutrients Digested...	3323	215	1079	42·7	100	1885	3223
Coefficients of Digestibility.....	59·5	54·3	56·5	39·5	40·8	64·5	60·3
SHEEP No. 38243 (7).							
Nutrients Consumed..	5586	396	1916	108·2	245	2920	5341
Excrement...........	2585	245·5	981	59·4	176	1120	2409
Nutrients Digested...	3001	150·5	934	48·8	69	1800	2932
Coefficients of Digestibility.............	53·8	38·0	48·8	44·9	28·2	61·6	54·9
Average (for 3 Sheep)	56·6	48·3	52·6	38·9	36·3	63·3	57·8

TABLE II—*(continued)*.

Coefficients: Digestibility of Hay—Panicum makarikari *(Superphosphate)*.

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL V (*a*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38240 (4).							
Nutrients Consumed..	4478·4	322	1550	92·3	203	2310	4275·4
Excrement...........	2349·7	183	822	60·4	145·5	1130	2204·2
Nutrients Digested...	2128·7	139	728	31·9	57·5	1180	2071·2
Coefficients of Digestibility..............	47·5	43·4	46·9	34·6	28·4	51·0	48·4
SHEEP No. 38248 (6).							
Nutrients Consumed..	4478·4	322	1550	92·3	203	23·0	4275·4
Excrement...........	2447·1	171·5	886	63·0	154·5	1165	2292·6
Nutrients Digested...	2031·3	150·5	664	29·3	48·5	1145	1982·8
Coefficients of Digestibility..............	45·4	46·8	42·8	31·8	23·9	49·6	46·3
Average (for 2 Sheep)	46·5	45·1	44·9	33·2	26·1	50·3	47·3

Coefficients: Digestibility of Hay—Panicum makarikari *(Bloodmeal)*.

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL V (*b*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38245 (1).							
Nutrients Consumed..	4492·8	342	1580	92·5	211	2260	4281·8
Excrement...........	2338·9	177·5	834	55·6	143·5	1125	2195·4
Nutrients Digested...	2153·9	164·5	746	36·9	67·5	1135	2086·4
Coefficients of Digestibility..............	48·0	48·0	47·4	39·9	32·0	50·1	48·8
SHEEP No. 38249 (2).							
Nutrients Consumed..	4492·8	342	1580	92·5	211	2260	4281·8
Excrement...........	2381·6	178·5	862	56·7	157	1110	2224·6
Nutrients Digested...	2111·2	163·5	728	35·8	54	1150	2057·2
Coefficients of Digestibility..............	48·0	47·8	46·0	38·7	25·6	50·8	48·4
SHEEP No. 38238 (3).							
Nutrients Consumed..	4492·8	342	1580	92·5	211	2260	4281·8
Excrement...........	2365·2	189	852	56·2	156	1125	2209·2
Nutrients Digested...	2127·6	153	728	36·3	55	1135	2072·6
Coefficients of Digestibility..............	48·5	44·8	46·1	39·2	26·2	50·2	48·4
Average (for 3 Sheep)	48·1	46·8	46·5	39·2	28·0	50·4	48·5

TABLE II—*(continued).*

Coefficients: Digestibility of Hay—Panicum makarikari *(Bloodmeal and Superphosphate).*

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL V (*c*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38247 (8).							
Nutrients Consumed..	4474	340	1525	95·0	228	2285	4246
Excrement...........	2269	172·5	817	57·4	138·5	1110	2130·5
Nutrients Digested...	2205	167·5	708	37·6	89·5	1175	2115·5
Coefficients of Digestibility.............	49·3	49·2	46·4	39·6	39·3	51·4	49·8
SHEEP No. 38251 (9).							
Nutrients Consumed..	4474	340	1525	95·0	228	2285	4246
Excrement...........	2144	163	786	54·2	146	1000	1998
Nutrients Digested...	2330	177	739	40·8	82	1285	2248
Coefficients of Digestibility.............	52·0	52·0	48·4	43·0	36 0	56·2	52 9
Average (for 2 Sheep)	50·7	50·6	47·4	41·3	37·7	53·8	51·3

Coefficients: Digestibility of Hay—Panicum phragmatoides *(Control).*

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL VI (*a*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38245 (1).							
Nutrients Consumed..	4459·2	508	1520	84·2	285	2060	4174·2
Excrement...........	2018·8	198	670	39·4	174	938	1844·8
Nutrients Digested...	2440·4	310	850	44·8	111	1122	2329·4
Coefficients of Digestibility.............	54·7	61·0	56·0	53·2	39·0	54·4	55·8
SHEEP No. 38249 (2).							
Nutrients Consumed..	4439·2	508	1520	84·2	285	2060	4174·2
Excrement...........	2166	221	680	42·2	184	1040	1982
Nutrients Digested...	2293·2	287	840	42·0	101	1020	2192·2
Coefficients of Digestibility.............	51·4	56·5	55·3	49·9	35·5	49·3	52·5
Average (for 2 Sheep)	53·0	58·7	55·6	51·5	37·2	51·8	54·1

TABLE II—*(continued).*

Coefficients: Digestibility of Hay—Panicum phragmatoides *(Superphosphate).*

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL VI (*b*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
Sheep No. 38240 (4).							
Nutrients Consumed..	4468·8	506	1540	77·5	269	2090	4199·8
Excrement...........	2309·1	205·5	792	41·1	175·5	1095	2133·6
Nutrients Digested...	2159·7	300·5	748	36·4	93·5	995	2066·2
Coefficients of Digestibility..............	48·3	59·4	48·5	47·4	34·8	47·6	49·2
Sheep No. 38247 (8).							
Nutrients Consumed..	4468·8	506	1540	77·5	269	2090	4199·8
Excrement...........	1926·7	185	605	34·1	150·5	950	1776·2
Nutrients Digested...	2542·1	321	935	43·4	118·5	1140	2423·6
Coefficients of Digestibility..............	56·8	63·5	60·7	56·1	44·0	54·7	57·7
Average (for 2 Sheep)	52·5	61·4	54·6	51·7	39·4	51·1	53·4

Coefficients: Digestibility of Hay—Panicum phragmatoides *(Ammonium Sulphate).*

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL VI (*c*).	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
Sheep No. 32839 (3).							
Nutrients Consumed..	4468·8	572	1510	76·2	286	2025	4182·8
Excrement...........	2164·1	260	645	37·5	184	1040	1980·1
Nutrients Digested...	2304·7	312	865	38·7	102	985	2202·7
Coefficients of Digestibility..............	51·6	54·5	57·2	50·8	35·8	48·6	52·6
Sheep No. 38236 (5).							
Nutrients Consumed..	4468·8	572	1510	76·2	286	2025	4182·8
Excrement...........	1869·9	234	550	32·7	164·5	892	1705·4
Nutrients Digested...	2598·9	338	960	43·5	121·5	1133	2477·4
Coefficients of Digestibility..............	58·0	59·0	63·6	57·1	42·6	56·0	59·2
Average (for 2 Sheep)	54·8	56·7	60·4	54·0	39·2	52·3	55·9

TABLE II—*(continued).*

Coefficients: Digestibility of Lucerne Hay.

	Absolute Dry Matter.	Crude Protein.	Crude Fibre.	Ether Soluble Extract.	Total Ash.	N. Free Extractives.	Organic Matter.
TRIAL VII.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
SHEEP No. 38248 (6).							
Nutrients Consumed..	4473	652	1710	100·0	389	1625	4084
Excrement...........	1988·4	193	914	57·6	171	655	1817·4
Nutrients Digested...	2484·6	459	796	42·4	218	970	2266·6
Coefficients of Digestibility..............	55·5	70·4	46·5	42·4	56·0	59·7	55·5
SHEEP No. 38243 (7).							
Nutrients Consumed..	4473	652	1710	100·0	389	1625	4084
Excrement...........	2185	238	944	63·4	242	697	1943
Nutrients Digested...	2288	414	766	36·6	147	928	2141
Coefficients of Digestibility..............	51·2	63·4	44·7	36·6	37·8	56·5	52·4
SHEEP No. 38251 (9).							
Nutrients Consumed..	4473	652	1710	100·0	389	1625	4084
Excrement...........	2162·6	250·5	908	62·6	238	696	1924·6
Nutrients Digested...	2310·4	401·5	802	37·4	151	929	2159·4
Coefficients of Digestibility..............	51·7	61·6	46·9	37·4	38·8	57·1	52·8
SHEEP No. 38252 (10).							
Nutrients Consumed..	4473	652	1710	100·0	389	1625	4080
Excrement...........	1999·2	189	962	57·9	177·5	583	1821·7
Nutrients Digested...	2473·8	463	748	42·1	211·5	1042	2262·3
Coefficients of Digestibility..............	55·2	71·0	43·8	42·1	54·3	64·1	55·3
Average (for 4 Sheep)	53·4	66·6	45·5	39·6	46·7	59·3	54·0

TABLE III.

B. *The Nutritive Ratio.*

Type of Hay.	Plot Treatment.	N.R.
Lucerne A	—	1 : 4·7
Lucerne B	—	1 : 4·2
Digitaria Pretoria Large	Control	1 : 14·5
Digitaria Pretoria Large	Superphosphate	1 : 10·9
Digitaria Pretoria Large	Bloodmeal	1 : 9·5
Digitaria Pretoria Large	Bloodmeal plus Superphosphate	1 : 9·2
Woolly Finger (Digitaria Pentzii).	Control	1 : 9·3
Woolly Finger (Digitaria Pentzii).	Superphosphate	1 : 10·2
Woolly Finger (Digitaria Pentzii).	Bloodmeal	1 : 9·7
Woolly Finger (Digitaria Pentzii).	Bloodmeal plus Superphosphate	1 : 9·4
Panicum makarikari	Control	1 : 15·4
Panicum makarikari	Superphosphate	1 : 13·2
Panicum makarikari	Bloodmeal	1 : 12·3
Panicum makarikari	Bloodmeal plus Superphosphate	1 : 11·9
Panicum phragmatoides	Control	1 : 6·7
Panicum phragmatoides	Superphosphate	1 : 6·4
Panicum phragmatoides	Ammonium Sulphate	1 : 6·3

TABLE IV.

Body-weights of Wethers (in lb.).

Nos. of Sheep.	Trial 1.			Trial 2.			Trial 3.			Trial 4.			Trial 5.			Trial 6.		
	Before.	After.	Gain or Loss.	Before.	After.	Gain or Loss.	Before.	After.	Gain or Loss.	Before.	After.	Gain or Loss.	Before.	After.	Gain or Loss.	Before.	After.	Gain or Loss.
38245............	73	73	0	73	70	−3	73	74	+1	74	75	+1	78	79	+1	79	79	0
38249............	69	70	+1	68	66	−2	71	73	+2	72	73	+1	75	75	0	75	77	+2
38239............	63	63	0	62	58	−4	59	60	+1	60	62	+2	67	67	0	70	70	0
38240............	(73)	—	—	71	69	−2	66	66	0	73	73	0	76	79	+3	77	77	0
38236............	65	65	0	64	68	+4	75	74	−1	66	67	+1	69	68	−1	72	72	0
38248............	65	67	+2	65	64	−1	(67)	(66)	—	70	71	+1	74	73	−1	73	74	+1
38243............	70	71	+1	71	67	−4	70	70	0	73	75	+2	(78)	(77)	—	78	80	+2
38247............	(66)	—	—	65	63	−2	73	73	0	67	68	+1	68	70	+2	69	71	+2
38251............	73	72	−1	(70)	(65)	—	64	66	+2	74	75	+1	80	79	−1	77	81	+4
38252............	60	63	+3	61	59	−2	74	75	+1	68	68	0	(72)	(67)	—	74	74	0

Section VII.

Toxicology.

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Notes upon the Isolation of the Alkaloidal Constituent of the Drug "Channa" or "Kougoed" (*Mesembryanthemum anatomicum* and *M. tortuosum*).

By CLAUDE RIMINGTON and G. C. S. ROETS, Section of Toxicological Chemistry, Onderstepoort.

THE "Kougoed" of the Bushmen of Namaqualand is a preparation compounded of the dried, over-ground portions of the plants *Mesembryanthemum anatomicum and Mesembryanthemum tortuosum*. It is chewed and is said to exert a strongly narcotic action in many respects resembling that of cocaine (Watt and Breyer-Brandwyk, 1932. Juritz (1905) observed dilation of the pupil.

Zwicky (1914) examined the preparation and extracted a crude alkaloid which he named "Mesembrin". Neither the free base nor any of its derivatives were obtained in the crystalline state; however, on the grounds of elementary analyses, Zwicky put forward the formula $C_{16}H_{19}NO_4$ for the alkaloid. Certain colour reactions were described.

Since the alkaloids of the Aizoaceae have received very little attention, a reinvestigation of the "Kougoed" seemed highly desirable. Through the kindness of Dr. W. Jordan, in placing at our disposal about 1·5 kilos of a preparation consisting of the dried plants, secured by him recently in Namaqualand, we were enabled to make a preliminary study of the alkaloidal constituents of the drug and to obtain crystalline salts of Mesembrin which establish its correct formula as $C_{17}H_{23}NO_3$. The present paper is a preliminary note upon the isolation and properties of this substance.

ABSENCE OF VOLATILE ORGANIC BASES.

Since piperidine has been found to be a constituent of *Psilocaulon absimile*, another member of the family Aizoaceae, (Rimington 1934) an aqueous acid extract of Kougoed was rendered alkaline and steam distilled into acid. No piperidine was found in the distillate which neutralised only 0·7 c.c. of N alkali per 100 gm. of plant.

ISOLATION OF THE ALKALOID.

The finely ground drug was extracted with boiling ether and the alkaloid then removed by continuous extraction with boiling chloroform. The dark solution was shaken with decolourising charcoal and evaporated to dryness, the residue being stirred up with dilute hydrochloric acid and a quantity of tarry material rejected. The aqueous solution containing the hydrochloride was again treated with charcoal, evaporated to dryness, and chloroform added to the dry residue when the hydrochloride of the base passed easily into solution. The filtered liquid was poured into several volumes of petroleum ether which precipitated the crude alkaloidal salt as a somewhat hygroscopic, pale buff-coloured powder. Attempts to crystallise it were not successful neither was the free base, which is moderately soluble in water, obtained in the crystalline state, although a crystalline picrate and platinichloride were readily obtained and the methylated free base was also crystallised with ease. From the composition of these crystalline derivatives, that of the free base was deduced.

PRECIPITATION AND COLOUR REACTIONS.

An aqueous solution of the alkaloidal hydrochloride gave amorphous precipitates with all of the usual alkaloidal reagents. Millon's reagent produced a pale red colouration and diazobenzenesulphonic acid in sodium carbonate solution an intense orange red colour indicating the presence in the molecule of phenolic groups. The dry material was dissolved by concentrated sulphuric acid with production of a yellow-brown colour. Mandelin's reagent, (Vanadium-sulphuric acid gave a greenish-blue colour on warming.

PREPARATION OF DERIVATIVES.

No attempt was made to prepare an exhaustive series of alkaloidal salts; since the amount of material available was very limited, attention was paid particularly to the picrate and platinichloride.

Picrate.

Aqueous picric acid in excess was added to an aqueous solution of the hydrochloride and the amorphous precipitate removed, washed and dissolved in sufficient warm 60 per cent. alcohol. On cooling, a little tarry matter separated and was filtered off. The filtrate in the ice-chest yielded crystals of the picrate consisting of prisms aggregated into clumps or rosettes. It is insoluble in ether, sparingly soluble in water and sparingly soluble in hot benzene but does not separate very well from the latter solvent alone. Recrystallisation was effected by adding excess of ether to a hot benzene solution and allowing the mixture to stand for some days. M.P. 193-4°.

*Microanalysis :**

		C	H	N
	Found	53·54	5·13	10·71
$C_{17}H_{23}NO_3 + C_6H_3N_3O_7$....................	**requires**	53·28	5·02	10·81

---

* Microanalyses by Dr. Backeberg of the University of the Witwatersrand, to whom we wish to express our thanks.

Picrolonate.

Prepared by the addition of aqueous picrolonic acid to a solution of the hydrochloride picrolonate is an amorphous yellow precipitate soluble in warm dilute alcohol but not crystallising readily. The material analysed contained some white ash which was allowed for in the calculations.

Microanalysis:

		C	H	N
	Found	57·82	5·76	12·02
$C_{17}H_{23}NO_3 + C_{10}H_8N_4O_5$....................	requires	58·56	5·64	12·66

Platinichloride.

Two forms of this salt were obtained. The amorphous precipitate produced when aqueous platinic chloride and alkaloidal hydrochloride are mixed separates, when crystallised from dilute alcoholic solution, in the form of small prisms having the melting point 151-3°, and containing neither water nor alcohol of crystallisation (see Fig. 1). On slow crystallisation in the ice-chest from aqueous alcohol, however, irregularly octagonal plates are obtained which contain two molecules of alcohol of crystallisation. Heated under the melting point microscope, softening is seen to occur with the evolution of bubbles at 170° but true melting does not take place before 181° is reached.

Microanalysis:

Salt with M.P. 181° dried in vacuo over P_2O_5 at 105°

	Per Cent.
Loss of weight..	8·03
Calculated for 2x C_2H_6O..	8·52

		C	H	N	Pt.
Above dried material.............	Found	41·20	4·84	3·12	20·48
Salt with M.P. 151-3′............	Found	40·52	4·95	3·33	19·64
$(C_{17}H_{23}NO_3)_2H_2PtCl_6$............	requires	41·28	4·90	2·83	19·74

A determination of methoxyl, CH_3O, and N-methyl,N-CH_3, carried out upon the crystalline platinum salt showed the presence of 2 methoxyl and 1 N-methyl group per molecule of base, thus:—

		CH_3O	CH_3 as N-CH_3
	Found	12·22	2·25
$(C_{17}H_{23}NO_3)_2H_2PtCl_6$ requires for 4 groups.............		12·54	3·03*

* For 2 groups.

Methylation.

Since colour reactions indicated the presence of a phenolic hydroxyl group, an attempt was made to obtain the fully methylated base by treating the alkaloidal hydrochloride dissolved in water with an excess of dimethyl sulphate and sodium hydroxide in the usual manner. The methylated derivative crystallised from the resulting reaction mixture in colourless four-sided prisms of M.P. 220-2° (see Fig. 2). The material so obtained contained a little ash which proved difficult to remove, therefore the platinichloride was prepared for analysis.

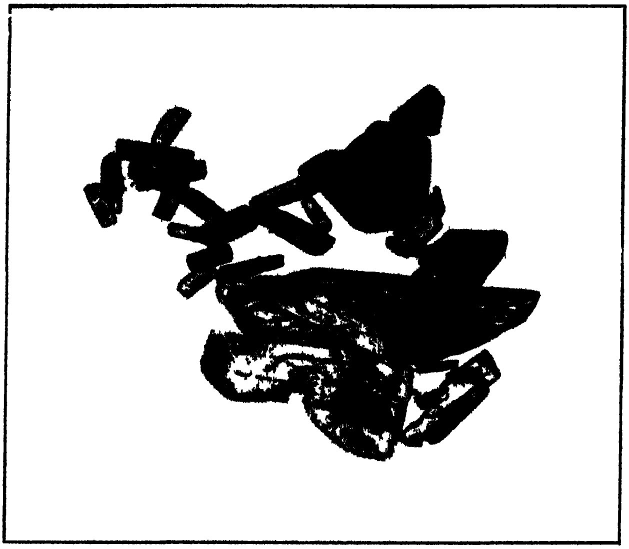

Fig 1 Mesembrin Platinichloride M P. 151-3° ×75

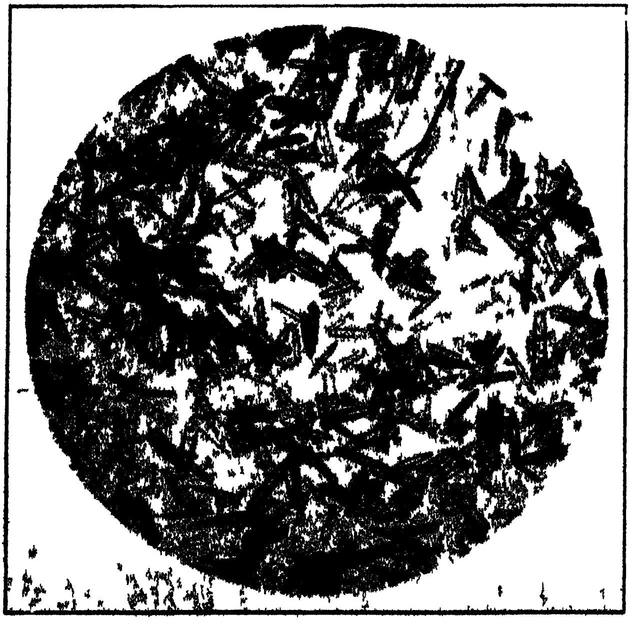

Fig. 2. Methyl-Mesembrin M.P 220-2°. ×85.

Microanalysis :

		Pt	CH_3O
	Found	18·72	18·85
$(C_{18}H_{25}NO_3)_2\ H_2PtCl_6$	requires	19·21	18·31*

* For 6 methoxyl groups.

An addition of one methoxyl group per molecule of base has thus been brought about. Methyl-Mesembrin gives a red colour with concentrated nitric acid and a crystalline precipitate with Wagner's reagent (Iodine in potassium iodide).

It would appear from the above considerations that the molecular formula of the alkaloid is $C_{17}H_{23}NO_3$ which can further be written

$$C_{14}H_{13}\begin{cases}-OH\\-OCH_3\\-OCH_3\\-NCH_3\end{cases}$$

It is of interest to compare this with the provisional formula put forward by Zwicky, namely $C_{16}H_{19}NO_4$. His material was impure and the error has fallen mainly upon the CH determination as might have been expected.

The molecular formula of Mesembrin, $C_{17}H_{23}NO_3$, is identical with that of Atropine and of Hyoscyamine. In view of the narcotic properties and reported mydriatic action possessed by Mesembrin, the question arises whether it may also belong to the series of Tropane alkaloids. Unfortunately very little material was available for the investigation of this point but a test hydrolysis was carried out upon a small quantity of the hydrochloride, using boiling saturated baryta as the hydrolytic agent, and the resulting reaction mixture separated definitely into an acidic fraction, crystallising in prismatic needles, and a basic fraction.*

The suggestion that Mesembrin belongs to the group of tropane ester alkaloids would thus appear to be strengthened.

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* We wish to thank Mr. P. Symons for carrying out this experiment.

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Chemical Investigation of the Plant *Acalypha indica*. Isolation of Triacetonamine, a Cyanogenetic Glucoside and Quebrachite.

By CLAUDE RIMINGTON AND G. C. S. ROETS, Section of Toxicological Chemistry, Onderstepoort.

The plant *Acalypha indica* (Euphorbiaceae), system No. 2730; 7/7/36, suspected of causing death of stock in the Potgietersrust district, was examined at this laboratory by Dr. D. G. Steyn and its toxicity established. Rabbits drenched with 12·5 gm. of the dry, powdered plant, succumbed within about two hours of dosing, exhibiting symptoms of dyspnoea and convulsions. The blood was found on post mortem to be dirty brown in colour. Hyperaemia of the gastric and duodenal mucous membranes was present. Natives of the district in which the plant grows employ it as an eye medicine under the name of "Machelikoane" but are unaware of its poisonous properties.

Tests showed the plant to contain cyanogenetic substances. A determination of the hydrogen cyanide liberated on maceration in buffer solution at pH 6·0 for varying periods of time was therefore carried out in the usual way; four to six hours maceration liberated the maximum quantity which corresponded to 268·2 mgm. HCN per 100 gm. of the powdered material on a dry weight basis.

Loss of moisture on drying sample: 6·6 per cent.

Time of maceration in hours.	HCN liberated from 5 gm. samples as c.c. of N/50 $AgNO_3$.
2·0	9·0
2·0	9·1
2·5	10·4
3·0	10·8
3·0	10·8
4·0	11·6
4·0	11·8
6·0	11·6
6·0	11·5
12·0	10·0
12·0	10·7
18·0	7·6
72	6·8

Maximum liberated = 11·6 × 1·08 × 20 mgm. HCN per cent.
= 250·5
On dry weight basis = 268·2

A determination of oxalate content was also carried out and afforded, in the mean, the figure of 3·14 gm. per 100 gm. dry material. Such a quantity could not account for the pathological symptoms of intoxication.

ISOLATION OF THE BASE TRIACETONAMINE.

During attempts to isolate the cyanogenetic glucoside, a substance was obtained which proved to be a salt of a base identified as triacetonamine, $C_9H_{17}NO$. The exact procedure undertaken was as follows:

The powdered plant material was extracted by cold 96 per cent. alcohol, the deep green extract decolourised by shaking with adsorbant charcoal, and evaporated to a syrupy consistency in an open dish at room temperature.

The residue was taken up in water, sufficient basic lead acetate added to cause complete precipitation then about 30 gm. of cadmium nitrate dissolved in water, and ammonia until addition caused no further turbidity. The cadmium nitrate was added since a cyclose derivative was found to be present and this reagent had been found useful for removing Pinit, inositol monomethyl ether from *Acacia* spp. in a previous investigation (Rimington, 1936). The filtrate from the lead-ammonia clarification was freed from excess of lead by hydrogen sulphide gas and evaporated to a syrup. Decolourising charcoal was then stirred in and drying continued by spreading the material in a thin layer in a vacuum desiccator until it could be powdered and introduced into a soxhlet extraction thimble for extraction by boiling ethyl acetate. This solvent on evaporation left a mixture from which a material crystallising in shining colourless plates was eventually isolated. These were sparingly soluble in absolute alcohol, did not possess any melting point but gave positive alkaloidal reactions, the precipitates with phosphotungstic acid and picric acid being crystalline; they also gave a crystalline 2:4 dinitrophenylhydrazone under the proper conditions with Brady's reagent. Of all these derivatives, only the picrate was found to possess a melting point; melting and decomposition occurred at 196°. Upon analysis, the crystals isolated were found to be the nitrate of the base $C_9H_{17}NO$. They gave a blue colour with the diphenylamine-sulphuric acid reagent, and the analyses of the other salts were in agreement with the formula as recorded below.

Nitrate.

Long flat plates with approximately parallel sides but oblique ends (see Fig. 1). On heating, some darkening occurred at about 163° and some sublimation at a higher temperature but no melting point was observed up to 350°. Sparingly soluble in hot acetone and easily recrystallised from this solvent by addition of petroleum ether.

*Microanalysis.**

		C	H	N
	Found	49·56	8·35	12·15
		49·43	8·34	—
$C_9H_{17}NO \cdot HNO_3$...........................	requires	49·53	8·26	12·84

Picrate.

By addition of aqueous picric acid and recrystallisation from hot dilute alcohol. Square-ended, flattened, golden prisms. M.P. 196° (see Fig. 2).

Microanalysis:

		C	H	N
	Found	47·14	5·51	14·68
$C_9H_{17}NO \cdot C_6H_3N_3O_7$.........................	requires	46·86	5·25	14·59

Phosphotungstate.

By addition of aqueous phosphotungstic acid. White precipitate, even in very high dilution, which on stirring crystallises in characteristically shaped crystals (see Fig. 3).

Platinichloride.

By addition of aqueous or alcoholic $PtCl_4$ to alcoholic solution; orange-yellow crystals easily recrystallised from hot water. No melting point.

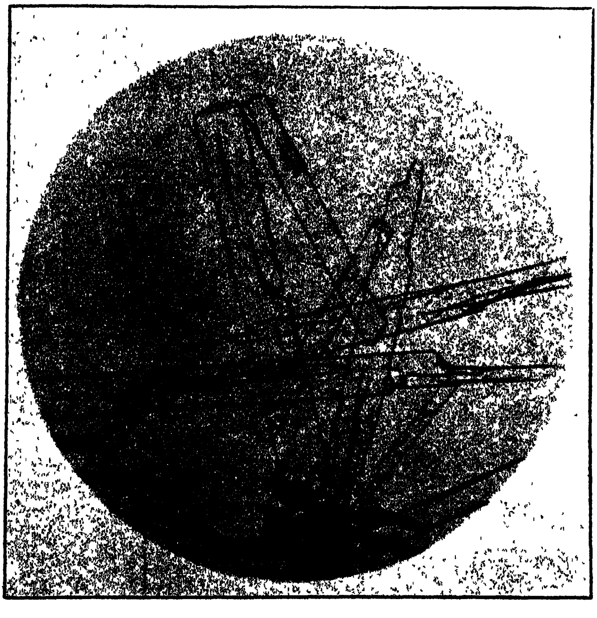

Fig. 1. Triacetonamine nitrate ex *Acalypha indica*, ×330.

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* Microanalyses by Dr. Backeberg of the University of the Witwatersrand to whom we wish to express our thanks.

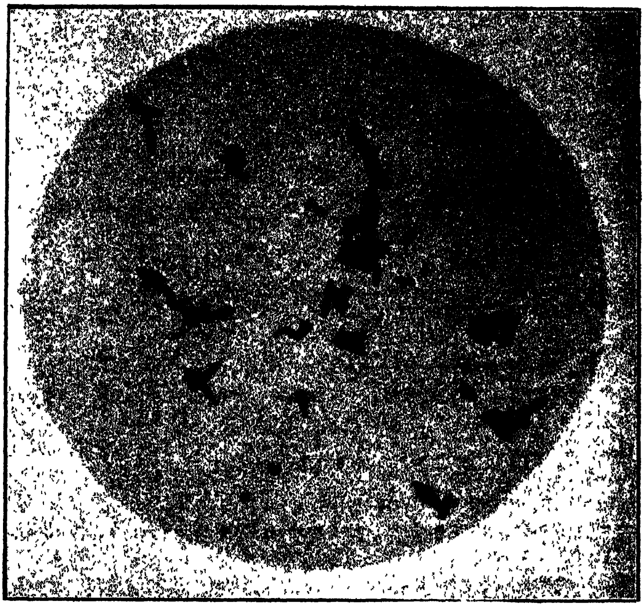


Fig. 2. Triacetonamine picrate (*Acalypha indica*), ×30.

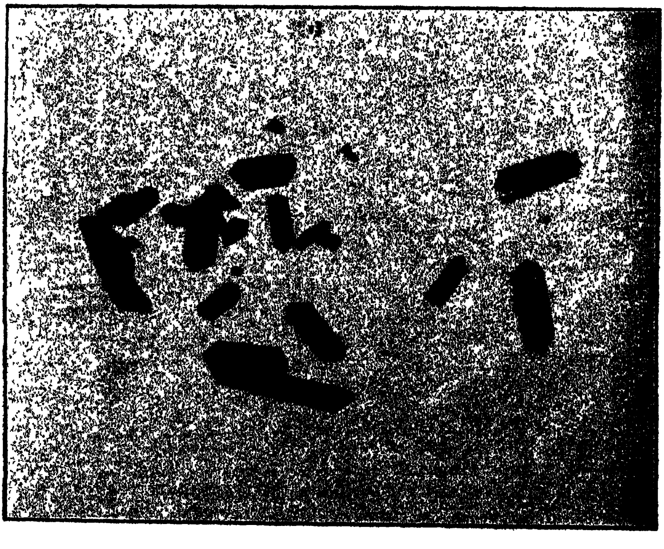


Fig. 3. Triacetonamine phosphotungstate, ×55.

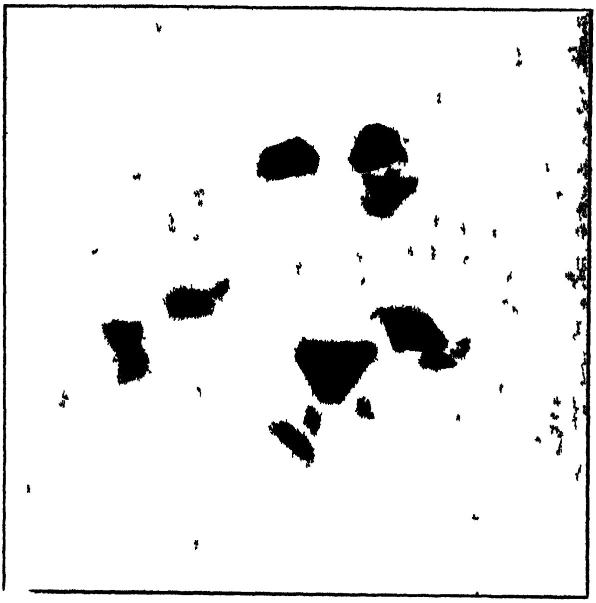


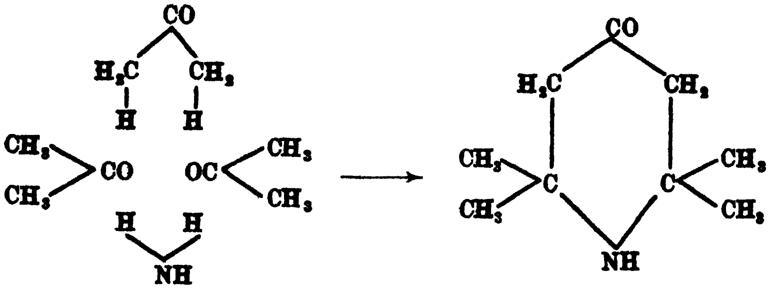
Fig. 4. Triacetonamine 2:4 dinitrophenylhydrazone ×66

2:4 Dinitrophenylhydrazone

By addition of Brady's reagent (solution of 2:4 dinitrophenyl hydrazine in 2N hydrochloric acid) to solution of crystals (B HNO$_3$) in 2N hydrochloric acid. Microcrystalline precipitate recrystallising from hot dilute alcohol in large yellow hexagonal tables usually resembling equilateral triangles with truncated apices (see Fig. 4). No melting point.

An examination of the literature suggested that the base closely resembled triacetonamine. This substance was therefore synthesised for comparison as described by Heinz (1874, 1875) by passing dry ammonia gas into boiling acetone for some hours, but the final isolation was accomplished not by way of the oxalate but by means of the well crystallised nitrate. The synthetic material was in every way identical with that isolated from *Acalypha indica* and the salts had identical crystalline forms. It is unfortunate that of all the salts of triacetonamine recorded in the literature, none has a true melting point. The picrate does not appear to have been prepared but as stated above the picrate of the isolated base melted at 196° and that of the synthetic material also had M.P. 196°, without depression on mixing. The characteristic 2:4 dinitrophenylhydrazone was also prepared crystallising in the large hexagonal tables described. To render the identification still further complete the oximes of the bases were prepared according to Harries (1896) who gives the M.P. of triacetonamine oxime as 152-3° (hexagonal prisms). 21.8 mgm. of the crystalline nitrate from *Acalypha indica* was dissolved in 0.3 c.c. of water, 6.9 mgm. of hydroxylamine hydrochloride and 0.2 c.c. of N sodium hydroxide added. The liquid was allowed to evaporate slowly at room temperature when a crop of large, shining, colourless hexagonal prisms separated. They were separated and pressed between filter papers and then had M.P. 150° yield 13 mgm. The free base was also prepared from a small quantity of the nitrate and found to have M.P. 56°. That of free triacetonamine plus H_2O is given as 58°.

The occurrence of such a substance as triacetonamine in a plant might at first sight occasion surprise but when one considers its possible formation, biologically as well as in vitro, from such abundant materials as acetone and ammonia it is seen to provide a beautiful example of the biosynthesis of a cyclic base from the simplest raw products, thus:



It must be pointed out at this stage that attempts to demonstrate the presence of basic material in the dry plant powder by means of extraction with Prolius' solution were unsuccessful, hence it is possible that the base was formed from some precursor during the working up of the material. Being a ketone, there is a reasonable possibility that it might enter into the composition of the cyanogenetic glucoside, which was found to contain other nitrogen than that combined as hydrogen cyanide, but this possibility will be no more than mentioned provisionally whilst the constitution of the glucoside remains obscure.

THE CYANOGENETIC GLUCOSIDE.

The isolation of the glucoside presents considerable difficulty occasioned firstly by the great instability of the material and secondly by its sparing solubility in ethyl acetate, a solvent otherwise very convenient for the isolation of glucosides.

Extracts of the plant material were made by percolation with cold 96 per cent. alcohol in some cases before and in others after a preliminary extraction with hot ether. The alcoholic liquids were shaken with decolourising charcoal and the solvent removed by fanning. Repeated extraction of the sticky residues with boiling ethyl acetate removed some glucoside. On cooling, the hot liquors deposited a mass of colourless crystalline material. This was freed from glucosidal impuritiy, taking advantage of its almost complete insolubility in hot absolute alcohol, recrystallised several times and identified as Quebrachite, a monomethyl ether of *l*-inositol, isomeric with Pinit.

It crystallised in large colourless prisms, M.P. 186°, and reduced ammoniacal silver nitrate on warming.

Microanalysis :

		C	H	N
	Found	43·10	7·32	nil
$C_7H_{14}O_6$....................................	requires	43·30	7·22	—

It was laevorotatory in aqueous solution.

Wt. = 72·4 mgm.
v = 10 c.c.
θ = −1·18°
d = 2

$$\therefore \left[\alpha\right]_D^{22} = \frac{-1\cdot18 \times 10 \times 100}{2 \times 7\cdot24}$$

$$= -81\cdot49°$$

Quebrachite has −80·6°.

The occurrence of Quebrachite in this euphorbiaceous plant is of interest. It occurs also in rubber latex (*Hevea brasiliensis, Euphorbiaceae*).

The mother liquors from which quebrachite had separated deposited on further concentration a rather sticky material rich in glucoside. Attempts to recrystallise from boiling ethyl acetate were not very successful but from hot acetone crystalline material separated in the form of fragile hexagonal plates. These had M.P. 182-4° and gave intense Molisch and hydrogen cyanide tests. The yield was very poor.

Attempts to improve the process of isolation met with very little success. Direct extraction of the plant with boiling ethyl acetate is not satisfactory; in spite of all precautions such as keeping calcium carbonate constantly present, a great deal of decomposition occurred with liberation of hydrogen cyanide.

The glucoside would appear to be dimorphic since an isomeric form was also encountered crystallising from ethyl acetate, on very slow evaporation, in the form of fine colourless needles, almost hair like and usually united in groups or bundles (see Fig.5). They had M.P. 108°.

Microanalysis :

		C	H	N
Glucoside M.P. 182–4°....................	Found	44·47	6·08	—
,, ,, ...	Found	45·86	5·77	7·69
,, ,, ...	Found	45·84	5·92	—
		44·49	6·08	6·91*
,, M.P. 108°....................	Found	44·32	6·51	7·98
$C_{14}H_{22}N_2O_{10}$....................	requires	44·42	5·87	7·40
$C_{14}H_{20}N_2O_{10}$....................	requires	44·67	5·34	7·45

* Trace of ash.

These figures would indicate a formula of either $C_{14}H_{22}N_2O_{10}$ or $C_{14}H_{20}N_2O_{10}$ but some uncertainty prevails owing to the fact that the analyses were not as strictly concordant as should have been the case had the different preparations been quite pure. The suggested molecular formulae are therefore put forward with all reserve but do suffice to show that the glucoside contains in its molecule more than one nitrogen atom. The high proportion of oxygen is also noteworthy.

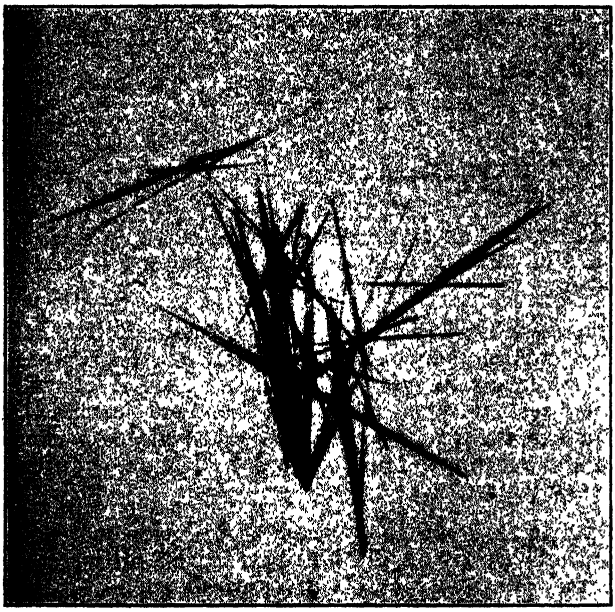


Fig. 5. Glucoside from *Acalypha indica*, M.P. 108°, ×30.

Hydrolysis experiments using emulsin or dilute sulphuric acid yielded hydrogen cyanide and dark, purplish coloured solutions from which no aglucone has as yet been isolated in a state of purity. It is noteworthy however that these hydrolysates give precipitates with alkaloidal reagents indicating the presence of some basic substance. The identification of this base must await the preparation of more pure starting material.

In one instance, Brady's reagent was added to a sulphuric acid hydrolysate in the hope of obtaining the dinitrophenylhydrazone of an aldehyde or ketone. A crystalline precipitate formed which when recrystallised from hot dilute alcohol had M.P. 187-9°. Analysis and mixed M.P. showed it to be identical with the 2:4 dinitrophenylhydrazide of acetic acid, the melting point of which is given as 187-190°. Both dissolved in aqueous sodium carbonate giving a brown solution.

Microanalysis :

		C	H	N
	Found	40·16	3·60	22·60
$C_8H_8O_3N_4$..................................	requires	39·98	3·36	23·34

Mixed M.P. with synthetic material 187–9°

The probability must be borne in mind, therefore, that one of the products of acid hydrolysis of the glucoside is acetic acid. This might conceivably arise from the hydrolysis of an N-acetyl group.

The above results are recorded with the object of putting on record the work already done and indicating the lines along which a more detailed investigation of the constitution of this unusual cyanogenetic glucoside might proceed.

SUMMARY.

(1) The plant *Acalypha indica* is cyanogenetic, containing approximately 270 mgm. HCN per 100 gm. dry weight of the dried powdered material.

(2) A base is present in extracts of the plant, being derived from some precursor, possibly the glucoside itself.

(3) This base has been identified as triacetonamine, the picrate (M.P. 196°) and 2:4 dinitrophenylhydrazone of which have been prepared and described.

(4) From the plant, Quebrachite, *l*-inositol monomethyl ether, has also been isolated.

(5) The cyanogenetic glucoside appears to crystallise in two forms, thin hexagonal plates, M.P. 182-4° and fine, silky needles, M.P. 108°. The molecular formula $C_{14}H_{20(22)}N_2O_{10}$ is suggested. It yields basic material on hydrolysis.

(6) From an acid hydrolysate of the glucoside, tested with Brady's reagent, acetic acid 2:4 dinitrophenylhydrazide has been isolated. The possibility of the presence in the molecule of an N-acetyl group is indicated.

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Section VIII.

Sex Physiology.

MALAN, A. I., MALAN, A. P., AND CURSON, H. H.	Studies in Sex Physiology No. 19. The influence of age on (*a*) amount and (*b*) nature and composition of the allantoic and amniotic fluids of the Merino ewe	205

Onderstepoort Journal of Veterinary Science and Animal Industry, Volume 9, Number 1, July, 1937.
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Studies in Sex Physiology No. 19.

The Influence of Age on (*a*) Amount and (*b*) Nature and Composition of the Allantoic and Amniotic Fluids of the Merino Ewe.

By A. I. MALAN, Section of Biochemistry and Nutrition, A. P. MALAN, Section of Statistics, and H. H. CURSON, Section of Anatomy, Onderstepoort.

(*a*) INTRODUCTION.

IN Study 18 (Malan and Curson, 1936) a few general remarks appeared on the nature of the increase in the amount of allantoic and amniotic fluids in the case of 10 Merino sheep, two having been slaughtered and examined at the end of each month of pregnancy. Whereas it seemed from Table I that the bulk of the increase of the *allantoic* fluid occurred during the second half of the gestation period, this does not appear to be the case when the data in Table 1 (below) are considered. In point of fact Table I contains additional details about nine sheep not included in Study 18. The variation is striking.

In the case of the *amniotic* fluid the amounts are not so extremely variable and there is apparently little increase before the end of the first month and after the end of the third month. The fluid therefore shows its increase during the second and third months.

OUR OBSERVATIONS.

The relevant information is tabulated in Table I.

TABLE I.

Foetus of ewe.	Foetus weight gm.	Allantoic fluid ccm.	Amniotic fluid ccm.	Age of foetus days.
35712	1	55	2·5	31
25924	0·9	48	2·5	31
35984	0·827	46	4	32
44803	1·25	56	6	33
44849	44·5	73	240	60
45082	54·2	95	210	60
35659	70·6	500	200	61
30169	59	195	250	61
35592	82	360	200	64
15337	530	76	520	90
21665	470	76	500	90
33131	617	300	200	92
39904	680	222	365	94
44679	2,730	690	930	121
38521	2,170	555	760	121
35976	2,230	350	450	122
44397	3,300	670	520	145
30514	3,540	1,140	300	146
45023	3,750	3,700	350	150

The data are represented graphically in Chart A where the amounts of the allantoic and amniotic fluids are shown by circles and black dots respectively.

A comparison with the chart (Fig. 493) figured by Needham (1931) shows more or less an agreement, but as there is so much variability it is not considered desirable to draw the curves. As is nevertheless evident, at the end of the first month the amniotic fluid is considerably less than the allantoic fluid, but then increases rapidly and at the end of the third month is apparently more than the allantoic fluid. Towards the end of pregnancy there is no obvious difference between the amounts of the two fluids.

When the amounts of the two fluids are compared with the foetus, as was done by Döderlein for the cow (Needham, Fig. 496) it is seen (Chart B), as pointed out by Needham, that both are much more abundant relative to the foetus weight during the early than in the late stages of pregnancy. This is so pronounced for the allantoic fluid that it has not been possible to include in the chart the values for the age of one month. The fall in the amount of the allantoic fluid per 100 gm. foetus weight with advancing age is much more rapid during the first three months than is the case with the amniotic, as may be expected in view of the less amount of the latter in the beginning, and its more rapid increase during the second and third months. This is demonstrated in Chart B, but it is to be noted that the curves shown are only rough illustrations of the situation and have no other meaning. Chart B is also in fair agreement with that drawn by Needham (Fig. 496).

CHART A.

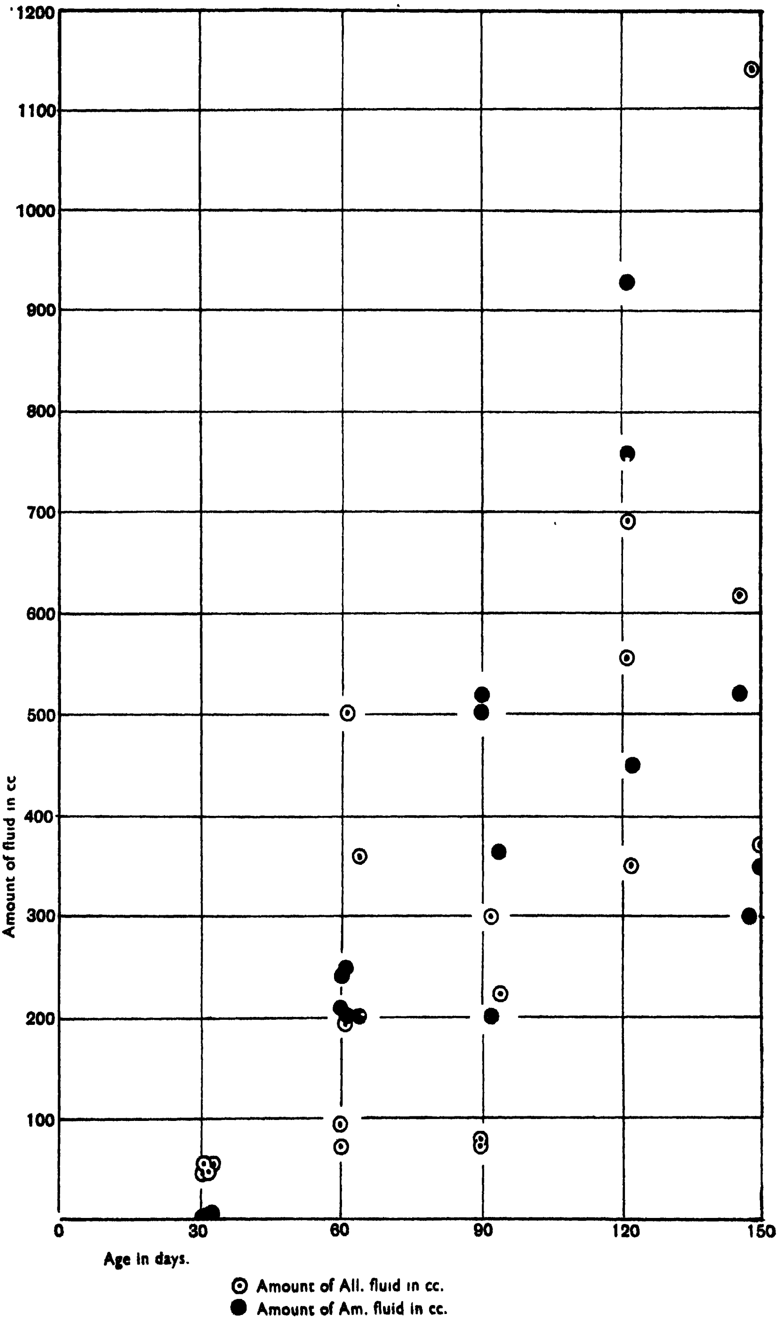
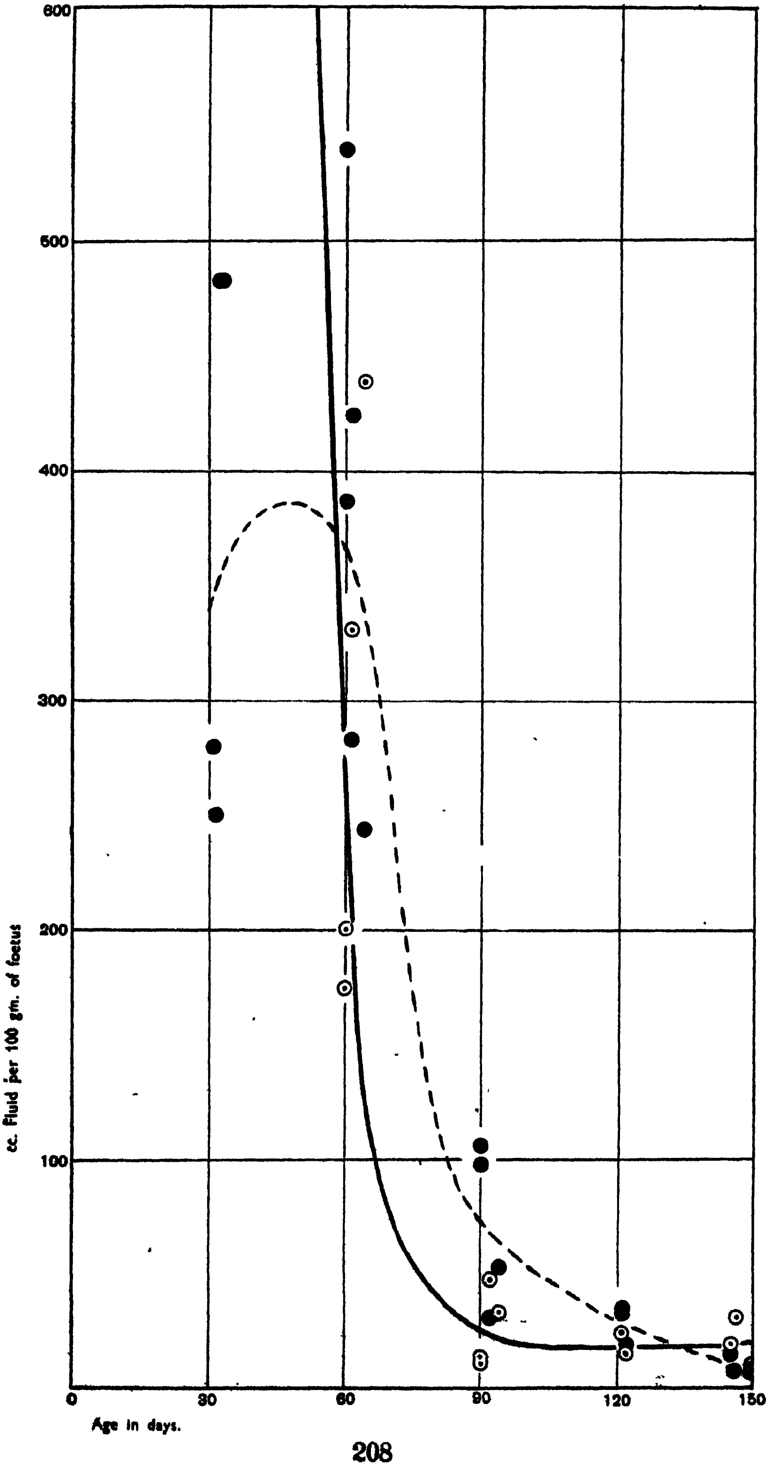

1200
1100
1000
900
800
700
600
500
400
300
200
100
Amount of fluid in cc
0
30
60
90
120
150
Age in days.
⊙ Amount of All. fluid in cc.
● Amount of Am. fluid in cc.

CHART B.


600
500
400
300
200
100
cc. Fluid per 100 gm. of foetus
0
30
60
90
120
150
Age in days.

(*b*) INTRODUCTION.

The nature and composition of the mammalian foetal liquids have been the subject of investigation for centuries and it is therefore surprising that so little precise information is available regarding them. Concerning the human, Tankard, Bagnall and Morris (1934) make the significant comment that " Although there are some analytical figures in the text books they are rarely of recent date and are also incomplete ". Although Needham (1931, p. 1534) indicates that the chemical study of foetal fluids offers " quick returns for work done " associated with " the economy of thought involved in trying any handy estimation method ", it is disappointing that so little data is available concerning the sheep. Needham in referring to this species mentions (*a*) Jacqué's work which indicates that " the allantoic fluid is at first hypotonic to the amniotic fluid and afterwards hypertonic to it ". Needham adds, " But as usual in this subject such a statement by no means applies to the pig or the cow, in which the allantoic liquid remains hypotonic to the amniotic liquid throughout development, the two approaching somewhat and reaching an almost equivalent osmotic concentration shortly before birth ". Again he concludes " one fact alone is universally true, namely that the ' foetal liquids ' are always a good deal hypotonic to the blood whether foetal or maternal ". (*b*) Needham, in Table 247, giving the analysis of the amniotic liquid *at term*, includes Jacqué's (1902) figures (in gm%) as follows:—total ash 0·84, soluble ash 0·82, insoluble ash 0·017, NaCl 0·64, and coagulable protein 0·102. (*c*) In regard to the carbohydrate content of the fluids, Paton, Watson and Kerr (1907) " reported the presence of fructose in " both the fluids of the sheep and cow. They " calculated the carbohydrate nitrogen ratio . . . and found the interesting, though rather enigmatic, result that C/N ratio diminishes in the allantoic as much as it is increased in the amniotic " (p. 1546); and (*d*) in discussing maternal transudation in foetal secretions Needham, in Table 249, gives an instructive comparison of an analysis of the fluids in the sheep and cow as follows:—

	Allantoic fluid.		Amniotic fluid.	
	Sheep.	Cow.	Sheep.	Cow.
gm. per cent.—				
Protein	0·54	0·08	—	—
Total ash	0·924	0·275	0·84	—
Insoluble ash	0·074	0·025	0·017	—
Soluble ash	0·85	0·25	0·82	—
NaCl	0·16	0·19	0·64	—
$\frac{\text{NaCl}}{\text{Soluble ash}} \times 100$	18·5	76·0	78·0	—

The precise age of the foetuses is not given.

Below are tabulated the more important facts relating to the nature of the foetal fluids of the ruminant and equine, very little precise data being recorded for the sheep.

TABLE II.

General Description of Ruminant and Equine Foetal Fluids(1).

	De Bruin. (1901.)	Smith. (1921.)	Craig. (1930.)	Williams. (1931.)
ALLANTOIC FLUID.				
Origin..............	Foetal urine.	Foetal urine.	—	Chiefly from foetal kidneys.
Colour..............	Whitish, colourless at first and later yellow or brownish.	First colourless but later brown.	First colourless but later becomes yellow.	—
Appearance..........	Foamy.	First turbid.	At first slightly turbid.	Clear.
Reaction............	Neutral.	—	—	—
Amount..............	Increases towards the end of gestation.	—	Greatest at early period.	Varies widely in health.
Specific gravity.......	Thin liquid.	—	—	—
Chemical composition.	Grape sugar, oxalates, albumen, mucus and allantoidin all absent.	Urea, allantoine (chemically related to uric acid), protein levulose, lactic acid and certain salts.	H_2O, albumen, osazone, urate of urea, lactic acid, phosphates of Na, Ca, Mg and traces of sugar. Later due to renal excretion are erythrin and hippuric acid. (Hippomanes contain oxalate of Ca.)	Albumen, sugar, and urea.
Function............	—	A second water jacket for foetus and at birth dilates vaginal passage.	— —	— —
AMNIOTIC FLUID.				
Origin..............	Secreted from internal face of amnion (2).	Transudate from both mother and foetus.	—	Not fully determined from amnion itself and or from foetal urine.
Colour..............	Amber.	First yellowish-red but reddish towards end, probably owing to meconium.	—	Colourelss but in foetal disease may be reddish or other colour.

TABLE II—*(continued)*.

	De Bruin. (1901.)	Smith. (1921.)	Craig. (1930.)	Williams. (1931.)
AMNIOTIC FLUID (*contd.*)				
Appearance..........	—	—	—	Clear but in foetal disease may be opaque.
Reaction.............	Alkaline.	Alkaline.	Alkaline.	—
Amount.............	—	—	Abundant and limpid during first half of gestation and later scantier, viscid and citron or red.	Sheep 100–500 gm. (quotes St. Cyr and Violet).
Specific gravity.......	Thin and watery but at about 5 months gestation changes into a mucoid stringy mass.	—	—	Slightly heavier than water.
Chemical composition.	H_2O 975–991 parts; chlorides. carbonates, calcium, potassium and sodium sulphates 2·6–7·8; urea 2–3 5; kreatin, kreatinin sugar and fat traces; albumen and mucin ·8–10·7.	Protein, urea, sugar, lactic acid, kreatin, and some salts.	99 per cent. H_2O, albumen, glucose, urea, kreatin and other elements of urine and meconium.	Albumen, sugar and urea (and contains bacteria). Foetal diarrhoea is observed in sheep.
Function............	Protection of foetus and lubrication of vaginal passage at birth.	Water bag for foetus and dilates and lubricates vaginal passage.	Equable temperature, protection and lubrication.	—

(1) As far back as 1814, J. F. John (in his *Chemische Tabellen* Needham, p. 222) shows that observations had been made on the foetal fluids of the cow. "In 1821 Lassaigne described the amniotic and allantoic liquids of the cow as containing 'albumen, osazone, a mucin containing nitrogen, amniotic acid, lactic acid, phosphate of calcium, sulphate of soda and potash and the muriates of soda and of ammonium'" (Needham).

(2) Zietschmann (p. 117) shares this opinion.

TABLE III.

An Analysis of the Foetal Liquids of the Merino Sheep at the end of the 1st, 2nd, 3rd, 4th and 5th Months.

D.O.B.	Days.	Fluid.	Total (c.c.).	pH.	S-G.	Gm. Solids per 100 c.c.	Gm. Organic matter per 100 c.c.	Gm. Ash per 100 c.c.	Gm. Nitrogen per 100 c.c.	M.gm. K per 100 c.c.	Mgm. Ca per 100 c.c.	Mgm. Na per 100 c.c.	Mgm. P per 100 c.c.
35712	31	Allantoic	55	—	1·003	0·82	·02	0·8	·087	20	8·24	223	5·95
		Amniotic	*2·5*	—	—	—	—	—	—	—	—	—	—
25924	31	Allantoic	45	—	1·005	1·06	·285	0·775	·063	30	6·54	254	5·95
		Amniotic	*2·5*	—	—	—	—	—	—	—	—	—	—
35984	32	Allantoic	46	8·4	1·004	1·15	·505	0·645	·061	12	11·8	204	3·45
		Amniotic	*4*	*8·6*	—	—	—	—	—	—	—	—	—
44803	33	Allantoic	56	8·2	1·003	1·10	·56	0·54	·061	19	11·5	182	3·8!
		Amniotic	*6*	*8·4*	—	—	—	—	—	—	—	—	—
44849	60	Allantoic	73	—	—	—	—	—	—	—	—	—	—
		Amniotic	*240*	*7·5*	*1·002*	*1·065*	*·2*	*0·865*	*·107*	*55·8*	*5·7*	*274·0*	*1·64*
45082	60	Allantoic	95	7·4	1·009	2·485	2·225	0·260	·239	9·8	9·5	58·8	2·07
		Amniotic	*210*	*7·6*	*1·003*	*1·05*	*·195*	*0·855*	*·051·*	*62·0*	*5·8*	*250·0*	*1·51*
35659	61	Allantoic	500	—	1·008	1·60	·85	0·75	·207	20·0	13·7	288·0	8·68
		Amniotic	*200*	*7·3*	*1·006*	*1·05*	*·435*	*0·615*	*·019*	*69·0*	*3·45*	*339·0*	*1·31*
30169	61	Allantoic	195	—	—	—	—	—	—	—	—	—	—
		Amniotic	*250*	*7·6*	*1·008*	*1·07*	*·195*	*0·875*	*·034*	*39·3*	*5·9*	*294·0*	*1·26*
35592	64	Allantoic Pregnant	360	7·3	1·009	1·49	·87	0·620	·138	6·0	15·7	239·0	7·94
		Allantoic Non-pregnant	—	7·3	1·007	1·47	·85	0·620	·140	6·0	16·5	224·0	8·2
		Amniotic	*200*	*7·3*	*1·008*	*1·09*	—	*1·200*	*·039*	*36·0*	*5·2*	*347·0*	*1·84*
15337	90	Allantoic Pregnant	39	6·6	1·018	4·23	3·37	0·86	·445	65·2	6·4	111·0	8·2
		Allantoic Non-pregnant	37	6·8	1·011	4·19	3·32	0·87	·426	30·8	4·8	117·6	11·3
		Amniotic	*520*	*8*	*1·007*	*1·27*	*·44*	*0·83*	*·109*	*45·3*	*5·9*	*242·5*	*1·49*

TABLE III—(*continued*).

D.O.B.	Days.	Fluid.		Total (c.c.).	pH.	S-G.	Gm. Solids per 100 c.c.	Gm. Organic matter per 100 c.c.	Gm. Ash per 100 c.c.	Gm. Nitrogen per 100 c.c.	M.gm. K per 100 c.c.	Mgm. Ca per 100 c.c.	Mgm. Na per 100 c.c.	Mgm. P per 100 c.c.
21665	90	Allantoic	Pregnant....	**48**	**6·8**	**1·02**	**5·30**	**4·49**	**0·81**	**·461**	**54·6**	**68·2**	**64·5**	**5·50**
			Non-pregnant	28	6·8	1·015	5·13	4·36	0·77	·432	24·4	33·9	64·5	5·0
		Amniotic..............		*500*	*7·4*	*1·008*	*1·31*	*·48*	*0·83*	*·074*	*30·8*	*9·2*	*247·0*	*0·68*
33131	92	Allantoic	Pregnant....	**300**	**6·7**	**1·010**	**2·65**	**2·18**	**0·470**	**·153**	**13·0**	**7·3**	**152·0**	**1·31**
			Non-pregnant	—	6·3	1·010	3·37	2·875	0·495	·248	—	6·1	33·0	1·16
		Amniotic..............		*200*	*7·8*	*1·004*	*1·28*	*·52*	*0·76*	*·031*	*26·0*	*7·8*	*290·0*	*1·5*
39904	94	Allantoic	Pregnant....	**140**	**6·0**	**1·013**	**3·950**	**2·990**	**0·960**	—	**123·0**	**14·6**	**141·0**	**18·73**
			Non-pregnant	82	6·0	1·014	3·815	2·825	0·990	·323	127·0	14·8	148·0	18·73
		Amniotic..............		*365*	*7*	*1·003*	*1·405*	*·595*	*0·810*	*·041*	*22·0*	*6·4*	*317·0*	*2·0*
44679	121	Allantoic	Pregnant....	**660**	**7**	**1·006**	**3·16**	**2·36**	**0·80**	**·195**	**42·0**	**12·9**	**174·0**	**9·78**
			Non-pregnant	30	7	1·016	2·98	2·23	0·75	·347	47·0	14·0	189·0	9·36
		Amniotic..............		*930*	*7*	*1·009*	*1·42*	*·73*	*0·69*	*·136*	*25·0*	*6·4*	*256·0*	*0·89*
38521	121	Allantoic..............		555	6·9	1·015	3·12	2·12	1·00	·277	225·0	9·1	131·0	3·45
		Amniotic..............		*760*	*6·9*	*1·006*	*1·38*	*·70*	*0·68*	*·161*	*25·0*	*6·8*	*244·0*	*1·057*
35976	122	Allantoic..............		350	6·9	1·016	4·3	3·705	0·595	·374	19·0	13·9	88·0	5·25
		Amniotic..............		*450*	*7·1*	*1·001*	*1·735*	*·985*	*0·750*	*·049*	*22·0*	*6·5*	*291·0*	*0·86*
44397	145	Allantoic..............		670	7·1	1·013	3·11	2·04	1·07	·245	407·0	5·9	14·0	4·64
		Amniotic..............		*520*	*7·2*	*1·005*	*1·51*	*·77*	*0·74*	*·091*	*134·0*	*5·7*	*178·5*	*2·85*
30514	146	Allantoic..............		1,140	7·4	1·004	2·77	1·85	0·92	·268	327·0	2·8	22·2	4·75
		Amniotic..............		*300*	*7·4*	*1·001*	*1·13*	*·405*	*0·725*	*·062*	*49·5*	*7·6*	*222·0*	*4·22*
45023	150	Allantoic	Pregnant....	**220**	**6·0**	**1·015**	**4·82**	**3·75**	**1·07**	**·416**	**246·0**	**16·4**	**66·7**	**12·5**
			Non-pregnant	150	6·3	1·014	4·35	3·3	1·05	·381	222·0	15·2	66·7	12·5
		Amniotic..............		*350*	*6·9*	*1·001*	*1·005*	*·185*	*0·82*	*·112*	*30·0*	*13·4*	*296·0*	*2·86*

DISCUSSION.

Here we will deal not only with the analysis of the amniotic fluid at full term (as was done by Needham) but also with the analysis of *both* fluids at the end of *each* month of pregnancy. As there appeared to be a difference macroscopically between the allantoic fluid of the pregnant and non-pregnant horns, samples of the respective liquids were examined from the second month onwards (2nd month, ewe 35592; 3rd month, all ewes; 4th month, 44679; and 5th month, 45023).

The specific gravity was estimated by the direct weight method and for the *entire* test, including chemical analysis, a minimum quantity of 30 c.cm. was essential. The gaps that appear in the accompanying Table III are in several cases due to insufficient fluid being available, e.g. the amniotic fluid at 1 month. Unfortunately no determinations were made regarding sugar, Mg or Al salts.

Although no hippomane was encountered in the allantoic fluid, a note has been added (see Appendix) regarding its nature since such structures are not uncommon in the sheep.

The results given in the above table (Table III) are unfortunately too incomplete to justify a detailed consideration of the chemical composition of the amniotic and allantoic liquids. Nevertheless, as the study reported in this publication was obviously of a preliminary nature it may be worth while to emphasize some of the indications suggested by the chemical data, especially in the light of the findings reported and discussed by Needham.

The values for hydrogen ion concentration, determined by the B.D.H. Capillator method, may not be indicative of the true values in every case as it was not always possible to carry out the determinations immediately after taking the samples. It would seem, however, that the values indicate greater alkalinity during the early part of gestation than at later stages, a fact which agrees with the observations made on hens' eggs by Gueylard and Portier and quoted by Needham. The pH of the amniotic fluid shows a tendency to be higher than that of the allantoic fluid of the same period as the values of Aggazzotti also indicate in the case of chickens. Determinations of the pH of foetal blood was omitted and is strongly indicated whenever possible in future work.

According to Needham hardly any investigations have been made of the specific gravity of the constituents of the hen's egg and apparently none of those of other terrestrial eggs. Groebbels has shown that the specific gravity decreases during the course of development of a number of wild birds' eggs. The values given in the table remain extraordinarily constant for both amniotic and allantoic fluids during gestation.

In regard to the total solids per 100 ml. fluid, it is evident that the allantoic liquid is significantly higher than the amniotic fluid and it would be interesting to make a complete partition of these solids. From the values of the other constituents given it is apparent that with the exception of sodium higher concentrations of the constituents are invariably present in the allantoic fluid.

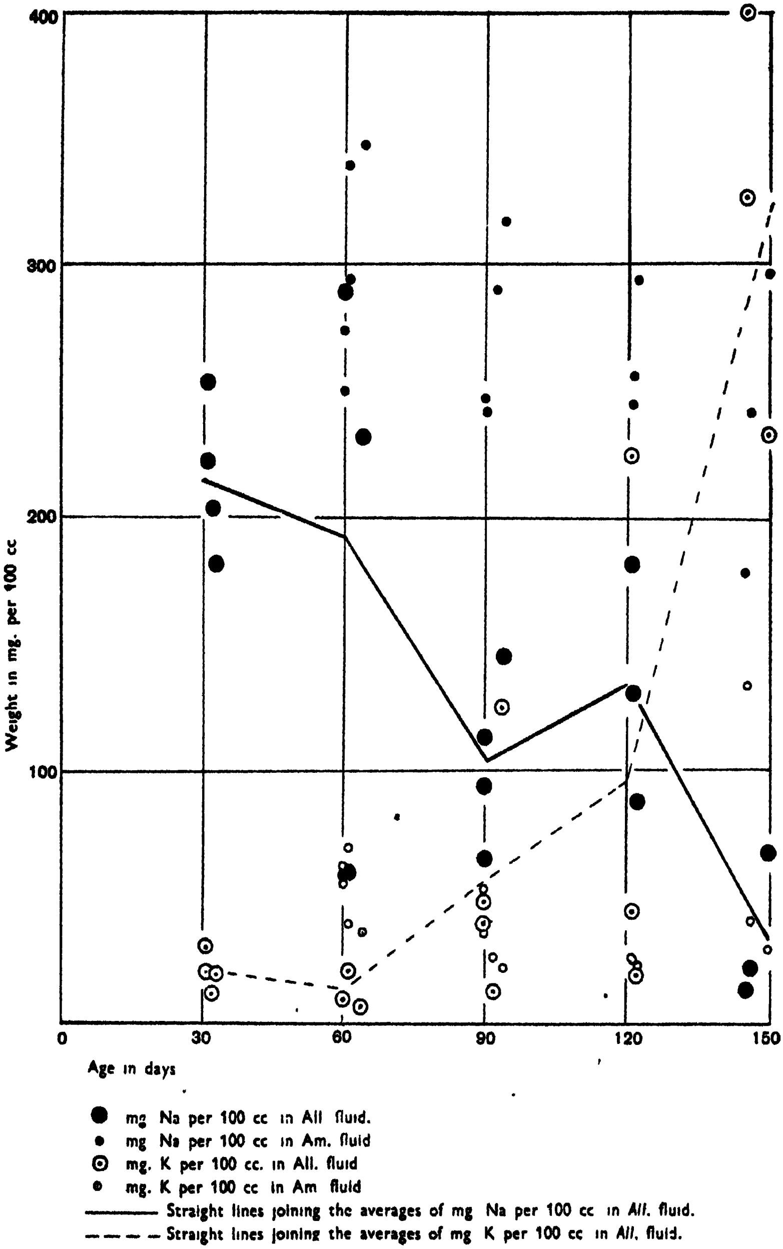
There is also a noticeable increase in the total solid content of the allantoic fluid round about the third month of gestation which is due in part to the formation of excretory products according to Needham. The values do not show a further rise after this period. The amniotic fluid apparently also increases in concentration after approximately three months. In the absence of mammalian amniotic and allantoic fluids given by other workers it may be mentioned that in the case of sheep there is by no means as marked an increase in total solids as in the chick embryo where approximately 29 times and 5 times the values obtained on the 9th day of incubation were obtained on the 14th day for the amniotic and allantoic liquids respectively. In the sheep's foetal fluids also the increase in total solids for both kinds of fluid was primarily due to an increase in the organic fraction of the fluids.

The total ash content of both the amniotic and the allantoic fluids fluctuates but remains fairly constant unless the increases registered in the allantoic fluid just prior to parturition indicate a higher tendency. A comparison with the results of Jacqué which are also indefinite suggests that further data should be collected on the ash contents of these fluids.

The total N content of the allantoic fluid is considerably higher than that of the amniotic fluid which supports Needham's statement on the origin of the fluid, viz.: the activity of the foetal kidneys and hence the depository of a proportion of the embryo's waste products. It would also seem that the concentration of N rapidly increases from the first to the second and third months of gestation. The N content of the amniotic fluid fluctuates very appreciably but apparently bears no direct relation to the N content of the allantoic liquid. However, a N partition is essential and is bound to contribute valuable data towards a discussion of the relative importance and significance of the presence of N fractions.

In regard to the remaining constituents which were determined, viz.: calcium, phosphorus, sodium and potassium, a comparison with the two values of each of these given by Needham according to Kamei for avian liquids is interesting. In the latter case it would appear that the Na values of both allantoic and amniotic liquids exceed the K values very considerably at both the periods of incubation given. This observation applies also to the amniotic fluids of the sheep's foetuses for all the months of gestation given in the table but not to allantoic fluids. In this case the Na content decreases as gestation advances while the reverse is true for K. The significance of this difference between the allantoic and amniotic fluids is not clear although the results of Groenewald on calf blood analysis may be quoted in this connection. At birth the K values—like those of the allantoic fluid—were well above normal while the Na values were well below normal for bovines. Both the increase of K and decrease of Na in the allantoic fluid during gestation appear to be too great to be unimportant for although the increase in K may be explained on the basis of the damming effect of excretory products containing K in the allantoic fluid the decrease in Na must clearly be explained on an entirely different basis. (See Chart C.)

CHART C.


400
300
200
100
Weight in mg. per 100 cc
0
30
60
90
120
150
Age in days
● mg Na per 100 cc in All fluid.
• mg Na per 100 cc in Am. fluid
⊙ mg. K per 100 cc. in All. fluid
∘ mg. K per 100 cc in Am fluid
——— Straight lines joining the averages of mg Na per 100 cc in *All*. fluid.
– – – – Straight lines joining the averages of mg K per 100 cc in *All*. fluid.

Phosphorus and calcium occur in greater concentrations in the allantoic than in the amniotic fluid and do not appear to be subject to regular increases or decreases during gestation although the fluctuations in the values are remarkable. Further work in view will probably provide more and therefore better data for discussing the significance of the values.

In conclusion attention should be drawn to the remarkable uniformity on the whole in the chemical composition of the allantoic fluids from the pregnant and non-pregnant horns of the uterus. The few differences in composition, although very considerable in some cases, must await the accumulation of additional data for their elucidation.

SUMMARY.

Sheep (Merino).

	Allantoic liquid.	Amniotic liquid.
Origin..........	"On the whole the evidence is decidedly in favour of *both* AM/L and AL/L in mammals being of foetal origin." (Needham, p. 1562.)	"Probably first a transudate and is afterwards added to by the foetal urine." (Needham, p. 1547.)
Colour..........	Varies from colourless waterlike fluid at end of first month, through a pale lemon at end of 2nd month, through orange at end of 4th month, to amber at full term.	Varies from colourless waterlike liquid at end of 1st month, through a pale lemon at end of 2nd month, through an orange at end of 3rd month, back to a pale lemon or even colourless liquid at full term.
Appearance.....	Varies from transparent or translucent appearance at end of first month to a translucent or opaque appearance from end of 2nd month onwards.	Varies from a transparent appearance up to the end of the 2nd month to a translucent at the end of the 3rd month, to an opaque and viscid fluid at the end of the 4th month and onwards.
Reaction........	More alkaline than amniotic liquid. See table 3.	—
Amount........	44 c.cm. (1 month), to 1,140 c.cm. (5 months). (Table 1.)	2·5 c.cm. (1 month), to 930 c.cm. (4 months). (Table 1.)
Specific gravity..	Values remarkably constant throughout gestation period.	See table 3.
Chemical composition	See table 3.	See table 3.
Function........	Protection of foetus and dilation of vaginal passage.	Protection of foetus and lubrication of vaginal passage.

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Thanks are due to Mr. J. G. Louw of the Chemistry Section who assisted in the various determinations and to Mr. F. D. Horwell of the Anatomy Section who gave valuable help in the collection of the fluids.

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APPENDIX.

A Note on the Hippomane.

Both Jenkinson (1913) and Zeitschmann (1923) describe the hippomane as a regular disc-like structure of varying size occurring in the equine, bovine and sheep. The origin is said to be due to an accumulation of embryotroph or uterine milk(³) which in some way collects in a fold of the allanto-chorion and becomes thrust inward (invaginated) towards the allantoic cavity. As indicated by the above authors the hippomane exists firstly as a vesicular structure, *i.e.* a diverticulum of the allanto-chorion containing embryotroph, secondly as a wart-like body attached by a stalk to the allanto-chorion (see Figs. 1 and 2) and thirdly as a free object in the allantoic cavity after rupture of the stalk. The density naturally varies according to age, the early structure being soft and composed of

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(³) Embryotroph is " an emulsion of cellular debris and some blood " (Arey, p. 86) added to " the increased glandular secretions (containing glycogan) " (Arey, p. 83) of the uterus, and which is absorbed directly by the trophoblast until the more efficient haemotropic type of nutrition has been developed.

cellular debris, blood and secretion from the surrounding uterine mucosa. The next stage would be represented by a firmer consistence owing to connective tissue proliferation. Finally, organisation would cease and the degenerating mass would become impregnated with salts derived from the allantoic fluid, *e.g.* ammonium magnesium phosphate, oxalic and uric acids, this being the case particularly in the equine. The colour varies from brown to olive green in the mare, a dirty white to orange in the cow, and a brown in the ewe. In the goat the colour is dirty white.

Zeitschmann (p. 151) states that in many stock-rearing centres it is believed that hippomanes if fed with salt on bread promote the secretion of milk.

Figs. 2-5 show hippomanes of the cow, donkey, sheep and goat (natural size).

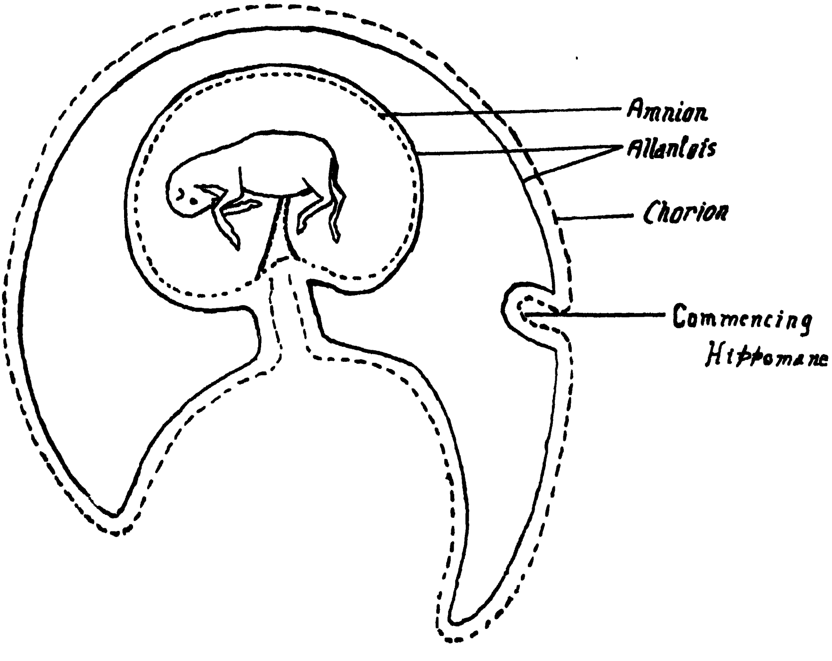

Fig. 1.

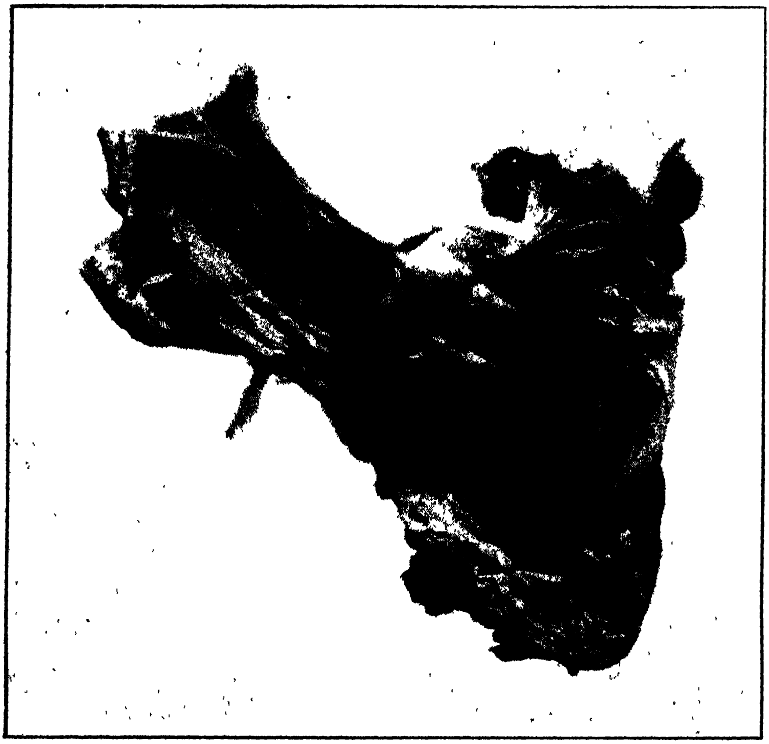

Fig. 2.

Fig. 3.

Fig 4

Fig 5

Figs. 4 and 5.

Section IX.

Photosensitisation.

RIMINGTON, C., QUIN, J. I., AND ROETS, G. C. S. Studies upon the photosensitisation of animals in South Africa X. The icterogenic factor in geeldikkop. Isolation of active principles from *Lippia rehmanni*, Pears 225

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Studies upon the Photosensitisation of Animals in South Africa.

X. The Icterogenic Factor in Geel-dikkop. Isolation of Active Principles from *Lippia rehmanni* Pears.

By CLAUDE RIMINGTON, Section of Toxicological Chemistry, J. I. QUIN, Section of Physiology, and G. C. S. ROETS, Section of Toxicological Chemistry, Onderstepoort.

In the seventh contribution to this series of articles dealing with photosensitisation, the naturally occurring disease of sheep, " Tribulosis " or "Geel-dikkop " was more particularly considered and it was shown (Rimington and Quin 1934) that the condition of photosensitisation arises as a result of the presence of the chlorophyll derivative, phylloerythrin, in the blood of sheep, this pigment having entered the circulation with the bile. The plant *Tribulus* presumably contains some toxic substance causing a well-marked icterus, the liver becoming incapable of eliminating bile and it is this hepatic disturbance which must be looked upon as fundamental to the development of the symptoms characteristic of the disease.

Since the experimental production of geel-dikkop by the feeding of *Tribulus* is rarely satisfactory, we have had recourse to the plant *Lippia rehmanni* (*Verbenaceae*), found by Quin (1933) to cause, when administered to sheep, an icterus indistinguishable from that present in geel-dikkop.

In an article by Rimington and Quin (1935) an account was given of preliminary experiments directed to the isolation of the active principle from *Lippia rehmanni*. The icterogenic activity of the plant was found to reside in an ether and alcohol soluble but water insoluble oleo-resin, and by fractionation of this material it was demonstrated that activity was confined to the portion soluble in 50-75 per cent. alcohol. By working up comparatively large quantities of the resin, a series of crystalline materials was isolated having acidic properties and certain of these were shown to be icterogenic when dosed to sheep. Finally, by an extension of the same technique of fractional crystallisation a material was isolated which was nitrogen free and to which the name " Icterogenin " was given. Two kinds of crystals were encountered both of which were active, the one consisting of prisms, M.P. 239°, the other irregular, fragile plates which melted at 155-160° but resolidified again to melt finally 230-6°. The preliminary change was associated with loss of weight and it was considered that alcohol of crystallisation was lost. Icterogenin when dosed in quantities of 1·5 gm. to a sheep *per os* caused distinct bilirubinaemia within 18 hours. Other physiological properties were also reported upon.

The method of isolation of Icterogenin left much to be desired. Fractional crystallisation could be expected, at best, to yield only a portion of the toxic principle actually present and the yields, moreover, were found to be variable. Since Icterogenin forms a sodium salt, attempts were made to better the procedure by shaking the crude ether extracts with aqueous sodium carbonate, but troublesome emulsions usually formed. On one occasion when salt had been added to break such an emulsion, the deposition was noticed in the liquid of a crystalline material which proved to be the sodium salt of Icterogenin and this observation provided a solution to the difficulties of isolation. Working over the sodium salt method we are now able to obtain quantitative isolation with perfect regularity and ease. A description of the technique is as follows.

ISOLATION OF THE ACTIVE PRINCIPLE IN THE FORM OF A SPARINGLY SOLUBLE SODIUM SALT.

The powdered plant material (500 gm.) is steeped in 96 per cent. alcohol (1,200 c.c.) overnight and after squeezing off in a press, the residue is subjected to a second similar extraction. The combined alcoholic extracts are shaken with sufficient decolourising charcoal (Kahlbaum, about 10 gm.) to remove all the chlorophyll, and the brown-yellow filtrate is fanned down to about ½ or ⅓ of its bulk at room temperature. An equal volume of ether is then added and much water, and the ether phase, which contains all the icterogenic material, is separated off in a large separatory funnel and washed with water. A small amount (15 to 20 c.c.) of 2·5 per cent. aqueous sodium carbonate solution is then added and, after shaking, the dark brown aqueous layer is removed. This first extract is worked up separately since, containing nearly all the dark pigments of the ether solution, the icterogenin obtained from it is not so pure as that from subsequent shakings.

When sufficient sodium carbonate solution has been used to remove all the Icterogenin from the ether phase, the combined alkaline liquids are again shaken once or twice with pure ether to free them from non-acidic substances. Finally, a layer of pure ether is left on the surface of the carbonate extract and then a few grams of sodium chloride stirred in. As the salt dissolves, the Icterogenin, if it be present in quantity, commences to separate in amorphous form at the interface. If the amount is small, crystalline sodium salt forms slowly. In any case, the mixture is left in an ice-chest until next day and the precipitate then filtered off through an ordinary paper filter and washed by ether. Further crops can usually be obtained by increasing the concentration of sodium chloride present and setting the mixture once more in the ice-chest. The precipitates are spread upon unglazed porcelain to dry and form light, felted masses, almost white in colour and often consisting of long needles, M.P. 228°, very sparingly soluble in water but readily soluble in alcohol. The yield from 1 kilogram of powdered *Lippia* was approximately 2·5 grams.

To prepare the free acid, the sodium salt was dissolved in warm alcohol, an excess of hydrochloric acid added and the solution diluted to make an alcohol concentration of about 60 per cent. On cooling, a mass of beautiful, shining white crystals fills the vessel. A specimen of this acid, which was positively icterogenic in the sheep test, when suspended in water and stirred with the calculated quantity of decinormal sodium hydroxide necessary for exact neutralisation dissolved almost entirely but the solution was not very stable, the addition of sodium salts such as normal saline or even long standing causing the slow separation of the sparingly soluble sodium salt of the active principle.

SEPARATION OF ISOMERS PRESENT IN THE CRUDE, FREE ACID.

Fractional crystallisation of the crude acid material obtained from the sodium salt showed that it was not homogeneous. In addition to the two types of crystals described in the previous communication (Rimington and Quin 1935), there was also obtained an acid crystallising in regular, narrow plates and melting at 161° without resolidification. It was just as active icterogenically and in the other pharmacological tests as were the substances originally described.

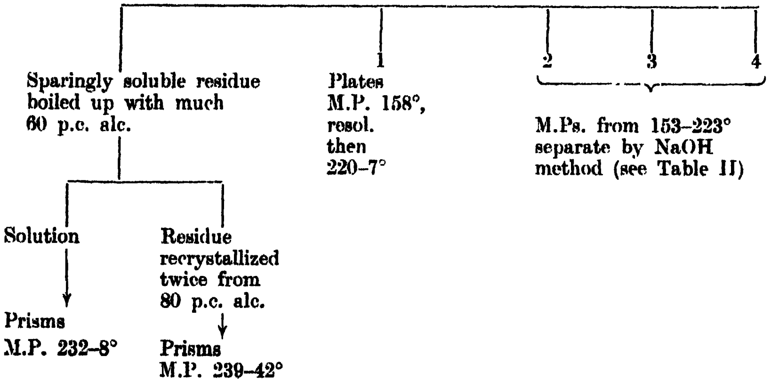
A re-examination of material prepared by the older method of direct crystallisation revealed the fact that by this method also small quantities of the new constituent were obtained, usually in admixture with the irregular, plate-like crystals.

Owing to their closely similar properties, separation of these individuals was not easily accomplished. The substance crystallising in small, elongated prisms and melting without preliminary change at 239° dissolves to an inappreciable extent when stirred with dilute alkali whereas the plate-like material goes readily into solution. A separation from the mixture of crude crystalline acids could thus be effected fairly easily but the substance on recrystallisation can, according to conditions, separate in the form of prisms or as irregular plates melting at 158° then resolidifying to melt finally at about the same temperature as the prisms namely 239°. This is strictly in accordance with the previous experience recorded by Rimington and Quin (1935) and the preliminary melt is associated with loss of weight. The third material, long regular plates, melting at 161° without resolidification, or loss of weight, is mose effectively obtained by fractional crystallisation. It dissolves readily in dilute aqueous alkali.

Attempts to utilise differences in solubility in salt solutions of the sodium salts of these acids for purposes of separation lead to a separation but only after a good deal of labour and the final application of fractional crystallisation. Differing solubility in dilute alcohol has proved to be most helpful as indicated in the accompanying Tables I and II.

TABLE I.

Crude material separating from dilute alcohol. Boil up with 4 successive lots of 60 per cent. alcohol.



Sparingly soluble residue boiled up with much 60 p.c. alc.

Solution → Prisms M.P. 232–8°

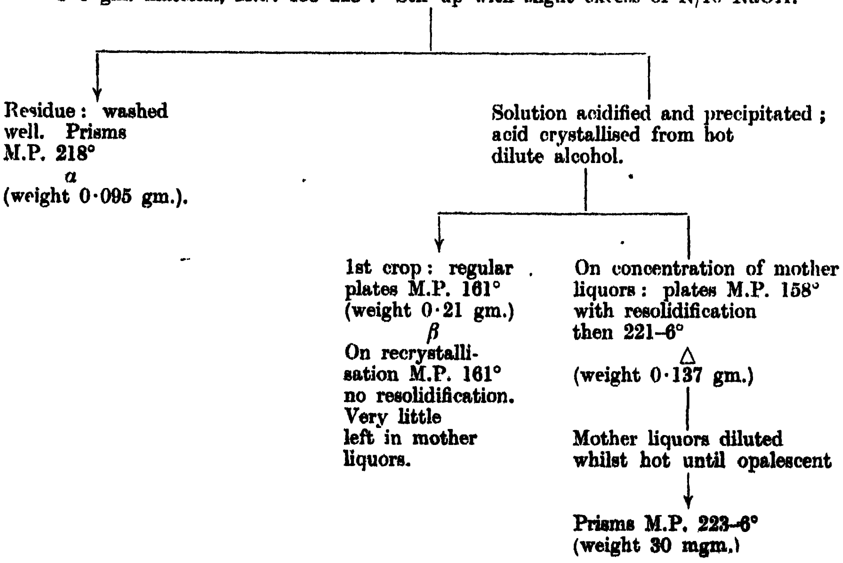
Residue recrystallized twice from 80 p.c. alc. → Prisms M.P. 239–42°

1 Plates M.P. 158°, resol. then 220–7°

2 3 4 M.Ps. from 153–223° separate by NaOH method (see Table II)

TABLE II.

0·5 gm. material, M.P. 153–223°. Stir up with slight excess of N/10 NaOH.



Residue: washed well. Prisms M.P. 218° α (weight 0·095 gm.).

Solution acidified and precipitated; acid crystallised from hot dilute alcohol.

1st crop: regular plates M.P. 161° (weight 0·21 gm.) β On recrystallisation M.P. 161° no resolidification. Very little left in mother liquors.

On concentration of mother liquors: plates M.P. 158° with resolidification then 221–6° Δ (weight 0·137 gm.)

Mother liquors diluted whilst hot until opalescent → Prisms M.P. 223–6° (weight 30 mgm.)

Repeated recrystallisation finally afforded three distinct crystalline fractions with fairly constant melting points:—

1. Prisms M.P. 239° (see Fig. 1) " Icterogenin A ".
2. Regular elongated plates M.P. 161° without loss of weight; no resolidification (see Fig. 2) " Icterogenin B ".
3. Irregular plates M.P. 158°, resolidifies to melt at about 230° (see Fig. 3) " Icterogenin C ". Apparently one molecule of water, H_2O, is lost at the lower temperature.

These three materials, which are all icterogenically active, gave on analysis the following figures. Several preparations were analysed.

*Micro-analysis.**

	C.	H.	M. Wt. (Rast)
Prisms, M.P. 239°. Icterogenin A...	73·65	9·17	525, 535
	73·85	9·44	634, 630
	73·69	9·32	
	73·94	9·23	544, 515
$C_{34}H_{52}O_6$ requires............	73·33	9·42	556·4
Plates, M.P. 161°, no resolidification, Icterogenin B....................	72·97	9·32	565, 530
	73·04	9·60	523, 506
	72·95	9·34	498, 494
	72·72	9·52	
	73·70	9·43	
Plates, M.P. 158° with resolidification. Icterogenin C..................	73·39	9·48	530, 525
	—	—	510, 500
	—	—	538, 570
After drying over P_2O_5 at 105° in vacuo..........................	75·44	9·64	Loss of wt. 2·55 per cent.
$C_{34}O_{50}O_5$....................	75·78	9·36	

The difficulty of obtaining satisfactory analytical figures from substances of the nature of these resinic acids is well known. Isomers are extremely difficult to separate and we still feel that some caution must be exercised in drawing a conclusion as to the molecular formulae of the materials here described. It would appear from a consideration of the analyses of the acetyl derivatives and of the 2:4 dinitrophenylhydrazones described below, taken in conjunction with the figures here reported for the free acids, that the formula $C_{34}H_{52}O_6$ is that best meeting all requirements.

The molecular weight demanded by such a compound is 556·4, a figure not far removed from the determinations made by Rast's method upon our various preparations. The camphor method is not one, however, from which very accurate results can be expected when the molecular weight is so large as is the case here. A confirmation of the molecular size is afforded by titration experiments and more particularly by the percentage of nitrogen present in the 2:4 dinitrophenylhydrazones. In some instances, figures greater than 600 were obtained by the camphor method when applied to prism preparations. This circumstance prompts us to observe that we do not deny the possibility of further isomers or closely related resinic acids occuring in our crude sodium salt derived from *Lippia rehmanni*. Further work will be undertaken in an effort to clear up these discrepancies and to place beyond all doubt the molecular formula $C_{31}H_{52}O_6$ which we now put forward for all three materials isolated.

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* Micro-analysis by Dr. A. Schoeller, Berlin.

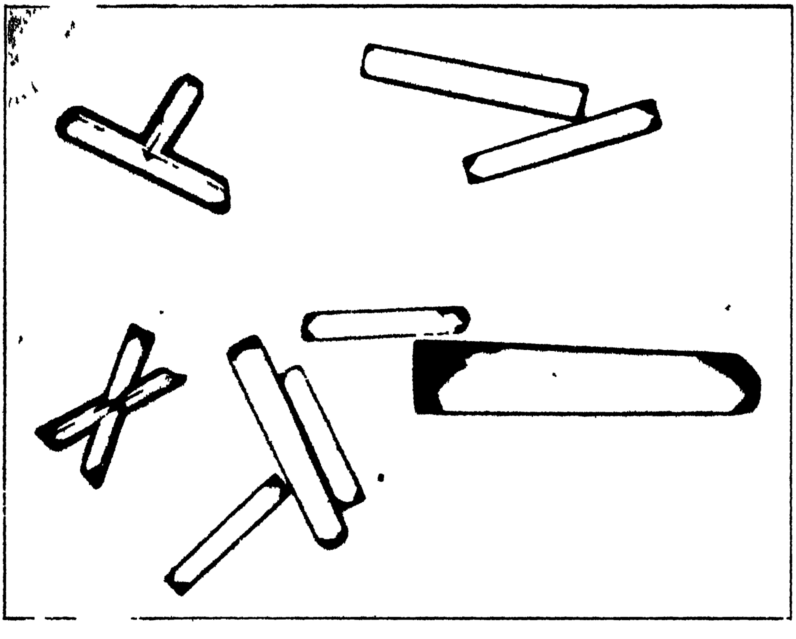


Fig. 1 Icterogenin A×300 Prisms M.P: 239°.

It would appear that the substances previously reported upon by Rimington and Quin (1935) were not quite pure and contained variable amounts of the third isomer which we now describe. For convenience and to avoid possible confusion at a later date, we propose to refer to the three active fractions we have now separated as Icterogenins A, B and C. All these materials are monobasic acids and possess, as will be shown later, one ketonic function. They give the Liebermann-Storch reaction with acetic anhydride and concentrated sulphuric acid, and a yellow colour with tetra-nitromethane, indicating the presence of ethylenic linkages. All are dextrorotatory to approximately equal extent thus precluding the use of rotation as an indication of purity.

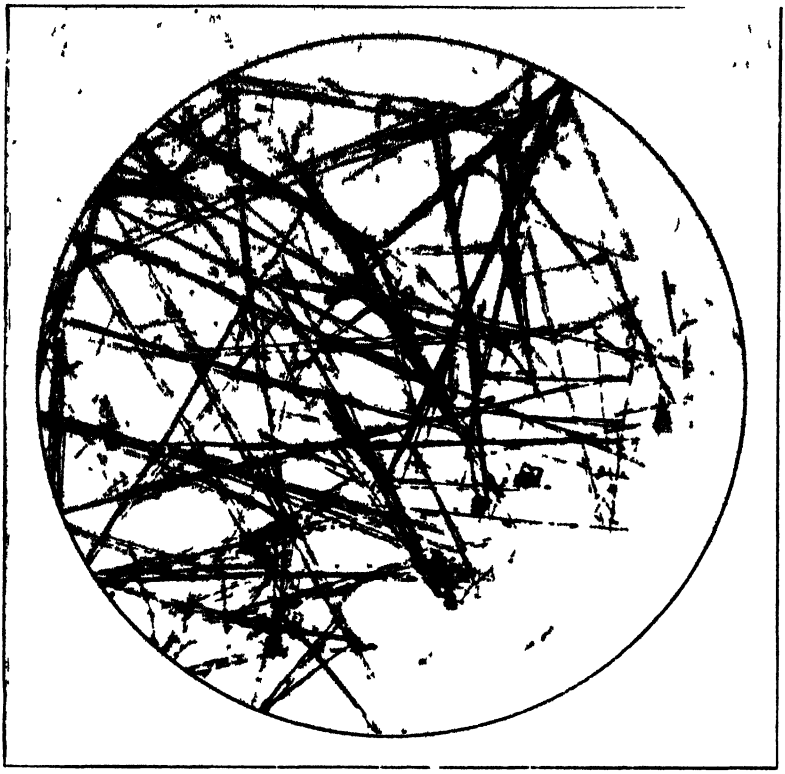


Fig. 2. Icterogenin B M.P. 161°×125.

OPTICAL ROTATORY POWER.

Icterogenin A. 50 mgm. in 15 c.c. of absolute alcohol in a 2 dm. tube was found to have a rotation of +0·48°

$$\therefore [\alpha]_{D}^{25} = \frac{+0\cdot48 \times 100 \times 15}{2 \times 5}$$
$$= +72\cdot0^{\circ}.$$

Icterogenin B. 50 mgm. of the plates M.P. 161° dissolved in 15 c.c. of alcohol had a rotation of +0·46°

$$\therefore [\alpha]_{D}^{28} = \frac{+0\cdot46 \times 100 \times 15}{2 \times 5}$$
$$= +69\cdot0^{\circ}.$$

Ictogernin C. 50 mgm. of the plates M.P. 158° and 230°, under similar conditions, had a rotation of +0·47

$$\therefore [\alpha]_{D}^{28} = \frac{+0\cdot47 \times 100 \times 15}{2 \times 5}$$
$$= +70\cdot5^{\circ}.$$

Micro-titration.

Icterogenin A. 35·0 mgm. of prisms were dissolved in 5 c.c. of an alcoholic KOH solution (5 c.c.≡3·88 c.c. of 0·09709 N HCl), a drop of phenolphthalein added, and the mixture immediately back-titrated.

Acid back 3·23 c.c.
Neutralised 0·631 c.c. N/10.
Theory for 1 acid group in $C_{34}H_{52}O_6$ 0·629 c.c. N/10.

A further 5 c.c. of alcoholic potash was added, the mixture refluxed for one hour and again back-titrated. It required 3·85 c.c. of acid indicating that no further acid groups develop under these conditions, i.e. the absence of lactone groupings. Similar results were obtained with all preparations.

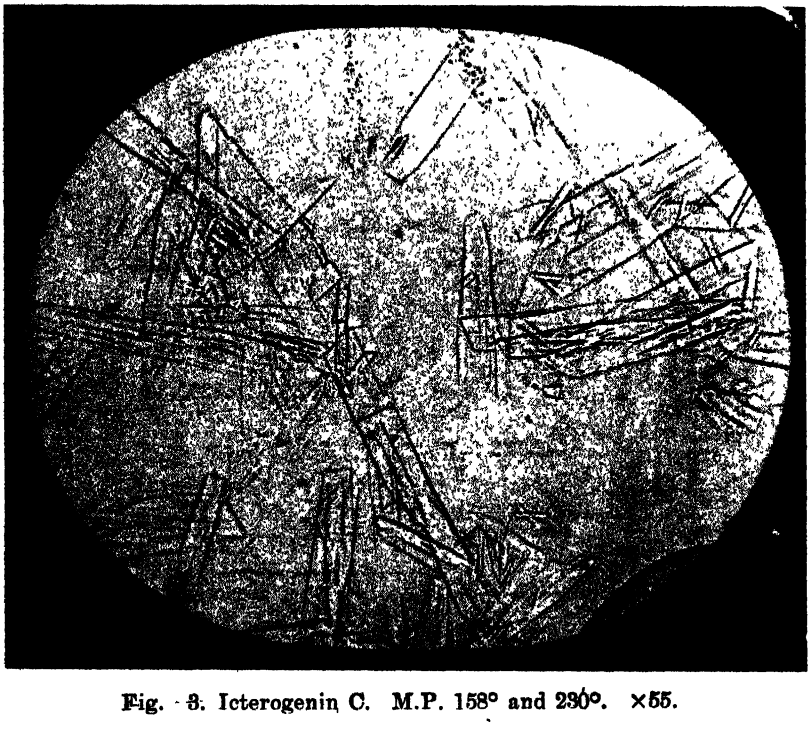


Fig. 3. Icterogenin C. M.P. 158° and 230°. ×55.

PREPARATION OF ACETYL DERIVATIVES.

In order to test for the presence of hydroxyl groups in the Icterogenins, the following experiments were carried out.

0·95 gm. Icterogenin A (prisms) was refluxed for 2·5 hours with 2·5 c.c. of acetic anhydride and 0·25 gm. of sodium acetate. The pale yellow mixture was poured into a quantity of ice water when a solid material separated. This was well washed and then crystallised from hot 80 per cent. alcohol. It separated in fine needles M.P. 140-41° Yield 66·4 mgm. (see Fig. 4).

Micro-analysis.

	C.	H.	M. Wt. (Rast).
Found	72·74	9·08	593, 587
$C_{34}H_{51}O_6(CH_3CO)$ requires	72·19	9·09	598·4

0·1 gm. Icterogenin B, similarly treated yielded an acetyl derivative crystallising in small prismatic needles M.P. 142°.

Micro-analysis.

	C.	H.	M. Wt. (Rast).
Found	72·25	8·87	569, 550
$C_{34}H_{51}O_6(CH_3CO)$ requires	72·19	9·09	598·4

Each substance yields a monoacetyl derivative indicating the presence in their molecules of one hydroxyl group. The melting points were so similar that a mixed melt was carried out and it was found that there was no depression (M.P. of mixture 140-2°). They are therefore identical and it can be concluded that Icterogenins A and B are true isomers; whatever constitutional difference there is that distinguishes them, it is removed during the process of acetylation.

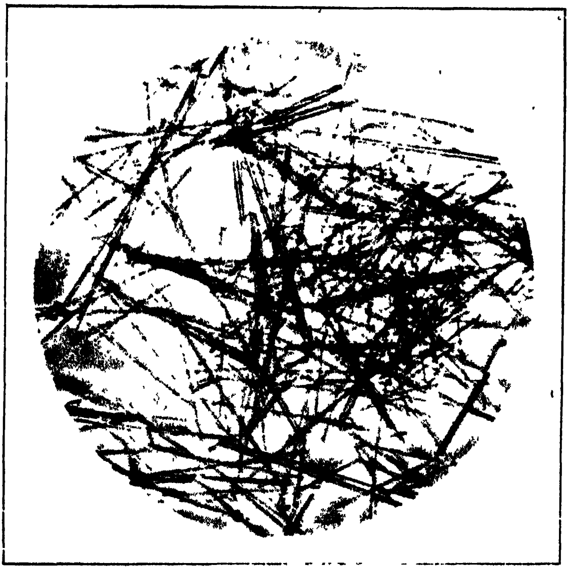


Fig. 4. Acetyl derivative of Icterogenin, M.P. 142°. ×135.

SAPONIFICATION OF THE ACETYL COMPOUNDS.

40 mgm. of acetyl-icterogenin A was dissolved in 5 c.c. of alcoholic KOH (5 c.c.≡3·88 c.c. of 0·09709 N HCl), a drop of phenolphthalein added and the mixture immediately back-titrated with the acid.

Acid back	3·12	c.c.
Neutralised	0·738	c.c. of N/10.
Theory for 1 free acid group	0·669	c.c. of N/10.

A further 5 c.c. of alkali was then added and, after refluxing for three hours, the mixture titrated as before.

Acid back 3·19 c.c.
Neutralised during saponification 0·670 c.c of N/10.

Theory for 1 acetyl group in $C_{36}H_{54}O_7$ 0·669 c.c. of N/10.

33·2 mgm. of acetyl-icterogenin B, similarly treated, afforded the following figures.

Titration before heating, neutralised 0·612 c.c. N/10.
Theory 0·555 c.c. N/10.
After 3½ hours saponification, neutralised 0·66 c.c. N/10.
Theory 0·555 c.c. N/10.

PREPARATION OF THE 2:4 DINITROPHENYLHYDRAZONES.

20 mgm. of each material was dissolved in sufficient dilute alcohol and, after acidification with 0·5 c.c. of concentrated hydrochloric acid, 1·5 c.c. of hot Brady's reagent was added. The precipitates which formed were centrifuged off, washed well with 2 N hydrochloric acid and crystallised from hot, dilute alcohol. The 2:4 dinitrophenylhydrazones which separated were orange-yellow in colour and had the following properties.

Icterogenin A 2:4 dinitrophenylhydrazone. Aggregates of fine needle-like prisms, M.P. 222-5°.

Derivative prepared from Icterogenin B. Aggregates of fine needle-like prisms, M.P. 226-9°.

Derivative prepared from Icterogenin C. Fine needle-like prisms, M.P. 224-8°.

Micro-analysis.

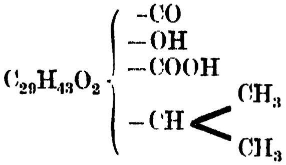
	C.	H.	N.
Derivative of Icterogenin A.........	63·74	7·50	7·44
,, ,, B.........	63·85	7·65	7·24
,, ,, C.........	63·65	7·55	7·09
$C_{34}H_{52}O_6 + C_6H_6N_4O_4$ requires...........	63·60	7·75	7·42

It will be noticed that allowance for elimination of H_2O is not made. We are at a loss at the present moment to explain why the derivatives should appear to possess H_2O more than is expected. Formulation of the parent substance with one more oxygen atom does not accord with analyses either of the free acids themselves or of their acetyl-derivatives.

DEMONSTRATION OF THE PRESENCE OF AN ISOPROPYL SIDE-CHAIN.

0·4 gm. of the sodium salt of Icterogenin was placed with 2 c.c. of acetic acid in a Pregl micro-kjeldahl distillation flask and, whilst passing a brisk current of steam through the apparatus, an oxidation mixture of chromic acid in glacial acetic acid was run in drop by drop as required. The distillate, which was collected in an ice-cooled receiver, was neutralised and again distilled, the first 10 c.c. being separately collected. To the distillate was added 2 c.c. of hot hydrochloric acid and 2 c.c. of hot Brady's reagent. The 2:4 dinitrophenylhydrazone which separated was well crystallised exhibiting the two types of crystals characteristic of the acetone derivative. It was centrifuged off, washed and recrystallised in the usual way and identified as acetone 2:4 dinitrophenylhydrazone.

The icterogenins therefore possess an iso-propyl side chain and the most probable general formula may consequently be written:



$$C_{29}H_{43}O_2\left\{\begin{array}{l}-CO\\-OH\\-COOH\\-CH<\begin{array}{l}CH_3\\CH_3\end{array}\end{array}\right.$$

The material remaining in the reaction flask after the oxidation was separated off, washed and recrystallised from hot dilute alcohol. It separated in clusters of white needles, M.P. 264-7°.

PHYSIOLOGICAL ACTION OF ICTEROGENIN.

The most characteristic action of icterogenin is the production of an intense and somewhat persistant bilirubinaemia without, however, post mortem signs of severe or extensive liver damage such as one would expect to accompany so severe a jaundice. Animals dosed with large quantities of *Lippia rehmanni* or with crude extracts prepared from the dried and powdered plant material invariably suffer from a very severe chronic constipation in addition to exhibiting bilirubinaemia and, after some days, clinical jaundice. These symptoms resemble closely those seen in natural cases of geeldikkop. If for some time prior to the dosing of *Lippia* extracts, a plentiful supply of green feed is provided, typical photosensitisation supervenes but not photosensitisation follows when the basal ration consists of bleached, dry straw devoid of chlorophyll. There would thus seem to be a very close similarity in every respect between *Lippia* poisoning and Geeldikkop provoked by feeding on *Tribulus*.

In article No. 9 of this series (Quin 1936) the physiological action upon the living animal of crude *Lippia* extracts and *Lippia* dosing has been fully discussed.

Experiments performed with the purified principles upon animals and upon isolated tissues are now recorded below.

Since it was found that these isomers exerted identical effects, the preparations used in the majority of experiments were obtained by decomposing the crude sodium salt and crystallising the resultant acid mixture from alcohol. For the effect of individual isomers, see experiments upon isolated intestinal strips.

DOSING OF ICTEROGENIN *per os*.

In numerous instances, quantities of Icterogenin or Icterogenin sodium salt varying between 1 and 4 grams were given to normal sheep *per os*. On occasions, the dose was preceded by 3 c.c. of 10 per cent copper sulphate solution in order to close the oesophageal groove and direct the material straight into the abomasum. In all cases, bilirubinaemia was noticeable within 24 hours and, with the higher doses, persisted for some days. A dose of 1 gm. is about the least quantity producing a distinctly positive bilirubinaemia in a 2 tooth sheep.

Where the jaundice was intense, it was usually noticed that on or about the third day after dosing there was present in the serum a small quantity of haemoglobin imparting to it an orange or brownish-yellow tint.

Following the oral administration of Icterogenin, a marked decrease in the number of ruminal movements per 5 minute interval was noticed and this effect persisted for some days (see below). In cases of animals with biliary fistulae, it was also observed that bile pigment rapidly disappeared and the bile eliminated had the characteristics of "white bile" as described in article No. 9 of this series.

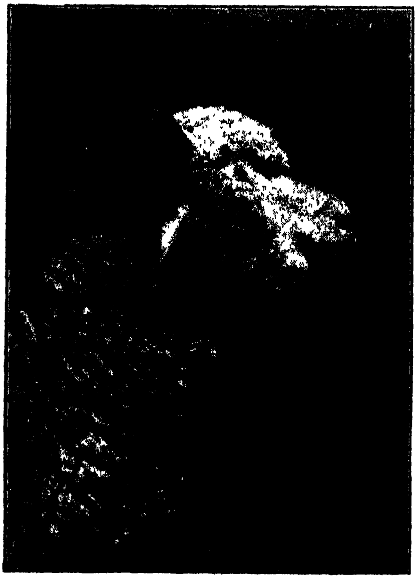


Fig. 5. Sheep, 48 hours after dosing with 2 gm. of Icterogenin per os.

The following experiment summarised below, illustrates these observations in a typical manner.

Sheep 44892 (age 7 months) was observed for 6 days prior to dosing, the ruminal movements being counted thrice daily and the faeces collected and weighed. The dose given was 2 grams of Icterogenin suspended in 250 c.c. of water and administered by stomach tube. Twenty four hours later, photosensitisation was apparent and increased in intensity as shown in the photograph Fig. 5 taken 48 hours after dosing. Even after 13 days, the serum remained slightly yellow but an eventual recovery took place with sloughing of the necrosed skin from the face and ears.

TABLE III. *Effect of oral administration of 2 gm. of Icterogenin.*

Date.	Ruminal movements per 5 minutes.			Wt. faeces per 24 hours. gm.	Serum.	Observations.
	a.m. 9	p.m. 1	4			
23/8/35	7	12	12	321 Normal......	Normal, water clear. Red ppt. 27 per cent.	Body wt. 42 lb.
24/8/35	7	10	—	360 Normal......	Normal.	
26/8/35	—	11	11	505 Normal......	Normal.	
27/8/35	5	12	11	462 sl. clumped..	Normal.	
28/8/35	7	12	10	312 Normal......	Normal.	
At 11 a.m. dosed 2 gm. Icterogenin by stomach tube.						
29/8/35	4	2	3	96 Soft..........	Yellow (++), clear. Red ppt. 26 per cent.	12 p.m. Restless, seeking shade, not feeding. 2 p.m. Definite swelling of face and ears.
30/8/35	4	0	2	230 badly formed	Darker yellow (+++), clear.	9 a.m. Swellings of face and ears, feet sore, yellow serum oozing from skin of nose. Photographed 4 p.m. Very badly swollen.
31/8/35	3	3	0	100 badly formed	As above, urine deep yellow	Swellings sl. subsided, fluid gravitating to intermandibular space. Sl. clinical jaundice. Kept from sun.
1/9/35	—	—	—	221 badly formed	As above........	Again badly swollen in sun.
2/9/35	2	7	8	61 badly formed, dry	As above........	Depressed. Necrosis of skin commencing. Feeding slightly.
3/9/35	5	8	12	230 pellets.......	As above........	Weight 35 lb.
4/9/35	6	9	10	232 pellets, dry..	As above........	Improving.
5/9/35	10	5	10	332 pellets, dry..	sl. lighter........	Improving.
6/9/35	5	9	8	313 pellets, dry..	sl. lighter........	Improving.
7/9/35	—	—	—	—	Light yellow.....	Improving.
9/9/35	—			—	Faint yellow.....	Clinical jaundice has disappeared. Skin sloughing. Improved.
10/9/35	—			—	—	Wt. 37 lb. Improvement rapid.
19/9/35	—			—	—	Animal discharged.

In the following case (see Table IV), a biliary fistula was introduced prior to dosing. The animal was eliminating approximately 260 c.c. of clear, dark green bile daily when it was given 4 gm. of Icterogenin by stomach tube. The subsequent appearance of the bile and serum is shown in the accompanying plate Fig. 6.

TABLE IV.

Date.	Bile.	Serum.	Obersvations.
30/7/35	246 cc. Deep greenish black	Water clear......	—
	10 a.m. dosed 4 g.m. Ictero-	genin by stomach	tube.
	4 p.m. No change in bile colour		
31/7/35	9 a.m. Very faintly greenish 4 p.m. Rather turbid. 404 cc. in all.	Definitely yellow.	Faint clinical jaundice.
1/8/35	380 cc. clear, very light yellowish	Deeper yellow....	Clinical jaundice. Photosensitive.
2/8/35	326 cc. pale, greenish yellow	Deep yellow.....	Not feeding.
3/8/35	256 cc. pale yellow........	Deep yellow.....	Losing condition.
4/8/35	246 cc. pale yellow........	Deep yellow.....	—
5/8/35	196 cc. greenish yellow....	Deep yellow.....	—
6/8/35	294 cc. pale yellowish......	Deep Yellow.....	Poor condition.
7/8/35	Found dead.		

The post mortem revealed the lesions typical of *Lippia* poisoning, including complete stasis of the fore-stomachs and severe stasis and putrefaction in the large intestine.

ADMINISTRATION OF ICTEROGENIN INTRAVENOUSLY.

Some difficulty attended the intravenous injection of Icterogenin on account of the insolubility of the free acid in water and the comparatively low solubility of the sodium salt. It would appear, however, that Icterogenin given in this manner is exceedingly toxic, possibly on account of the fact that it possesses haemolytic properties (see below). A sheep which received 0·5 gm. dissolved in a little 70 per cent. alcohol showed no ill effects apart from a slightly hurried respiration. The serum, however, became slightly, but distinctly icteric within 48 hours of dosing.

Upon increasing the dose to 1 gm., very severe toxic effects became apparent. A sheep injected intravenously with such a dose at 10.30 a.m. was found dead at 1 p.m. At post mortem it showed pronounced hyperaemia of the spleen, hyperaemia and slight degenerative changes in the liver, with oedema of the gall bladder, and intense hyperaemia of the mucosa of the small intestine together with haemorrhages into the lumen.

HAEMOLYTIC ACTION OF ICTEROGENIN.

The icterus of geel-dikkop and *Lippia* poisoning is not an haemolytic icterus, nevertheless Icterogenin does exhibit a fair degree of haemolytic activity in vitro. Most probably, in the living animal, the liver eliminates the toxin from the portal circulation as fast as it is absorbed and destroys or excretes it via the bile. Should the assault upon the liver be sufficiently great, however, the cells of this organ become thrown out of action to such an extent that elimination of bile becomes impaired or impossible and a vicious circle may then be set up, i.e. an obstructive or regurgitative jaundice supervenes.

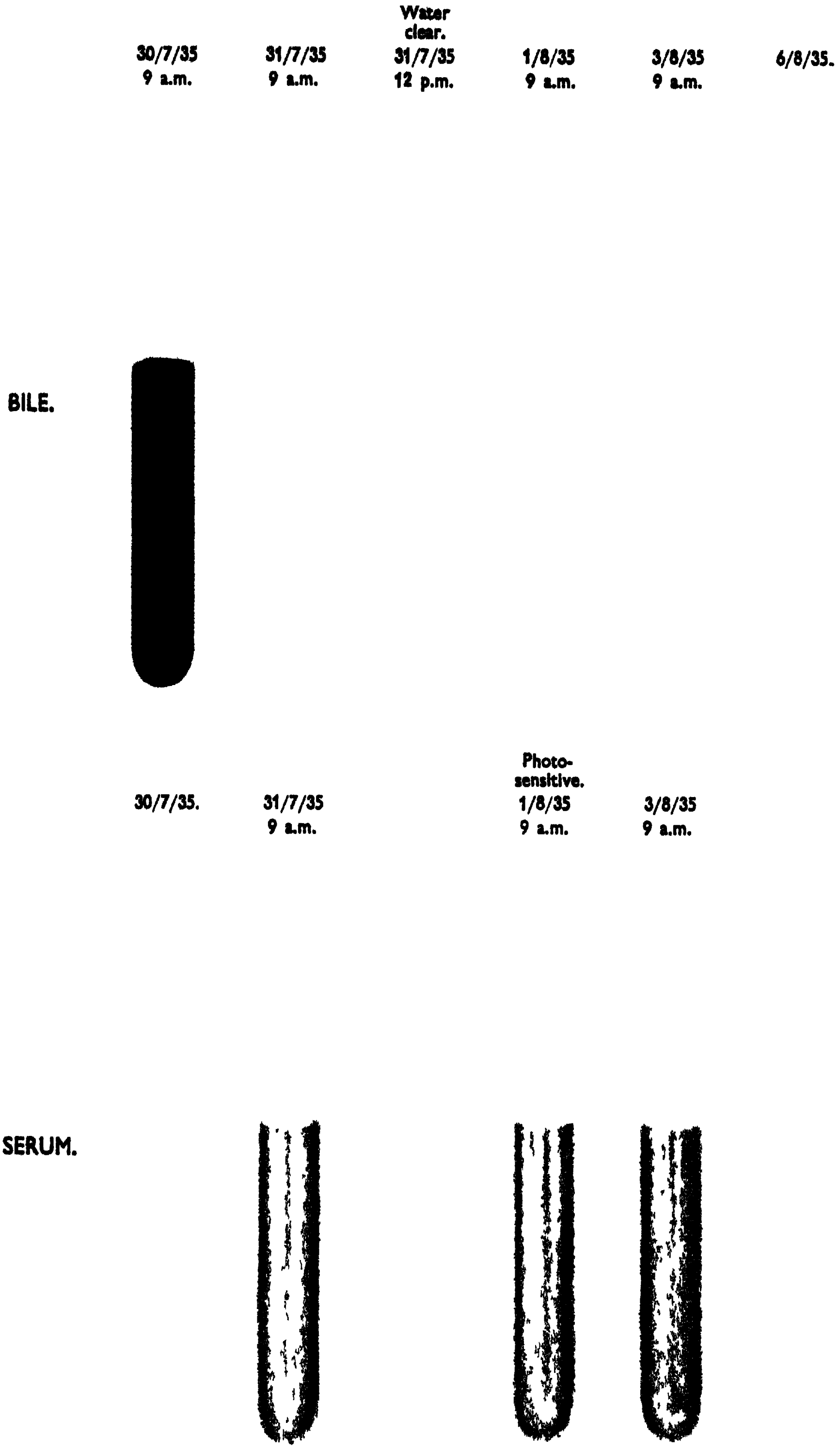


Fig. 6.—Fistula Bile and Serum of Sheep receiving 4 gm. Icterogenin *per os* at 10 a.m. on the 30/7/35.

The in vitro haemolytic action of Icterogenin was demonstrated as follows. A suspension of washed sheep's cells in Ringer-Locke solution was made up by diluting 0·2 c.c. of cells to 100 c.c. 1 c.c. aliquots of this suspension were measured into test-tubes. The Icterogenin solution consisted of 10 mgm. of pure substance dissolved by the addition of the requisite amount of alkali (0·18 c.c of N/10 NaOH) in a total volume of 2 c.c. 1 c.c. of this solution was diluted to 25 c.c. to prepare the stock solution which thus contained 0·2 mgm. per c.c. The volumes added to the blood suspension varied from 0·15 to 0·4 c.c. and suitable controls showed that no haemolysis occurred within half an hour after the addition of the latter quantity of water alone.

The results obtained are presented below and in the curves Figs. 7 and 8. It will be seen that Fig. 8 is constructed by plotting log 1000/*t* against log C × 10^5 where *t* is the time in seconds and C is the concentration of Icterogenin in grams per c.c. The points lie approximately upon the straight line $C^n t$ constant until the last two points where a large increase in the quantity of Icterogenin added made little difference to the time taken for complete haemolysis. Clark (1933) points out that the relationship $C^n t$ = constant holds also for the action of saponins investigated by Ponder and Yeager (1931), ammonia, etc. upon blood cells and it appears to us that the deviation from this relationship at relatively very high concentrations can probably be explained by the intrusion of a fresh limiting factor. For example, the regular portion may represent the rate of combination of drug with cell, the latter portion, where a large excess of drug has been added, the time taken for the cells to swell up and burst or some such similar mechanism.

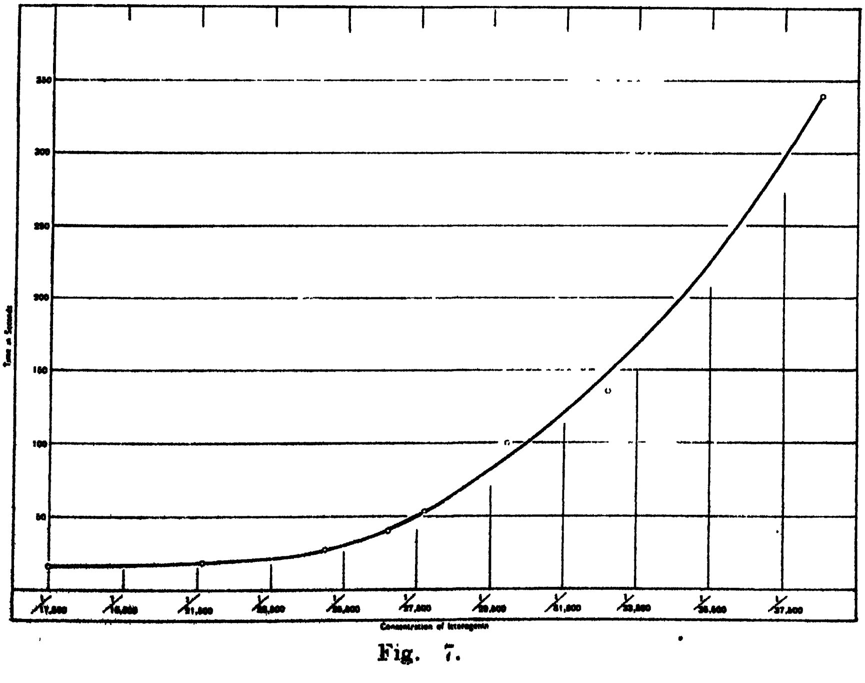


Fig. 7.

HAEMOLYTIC ACTIVITY OF ICTEROGENIN.

Vol. added. c.c.	*Quantity added.* mgm.	*Total volume.* c.c.	*Dilution.*	*Time for haemolysis.* Secs.		*Mean.* Secs.
0·4	0·08	1·4	1/17,500	15	16	15·5
0·3	0·06	1·3	1/21,667	17	19	18
0·25	0·05	1·25	1/25,000	29	26	27·5
0·23	0·046	1·23	1/26,740	40	40	40
0·22	0·044	1·22	1/27,730	55	52	53·5
0·20	0·04	1·20	1/30,000	100	100	100
0·18	0·036	1·18	1/32,780	136	136	136
0·15	0·03	1·15	1/38,333	346	334	340

Log 1000/t.	Log C x 10^5.
1·8097	0·7570
1·7447	0·6643
1·5607	0·6021
1·3979	0·5729
1·2716	0·5571
1·0000	0·5229
0·8665	0·4844
0·4685	·4164

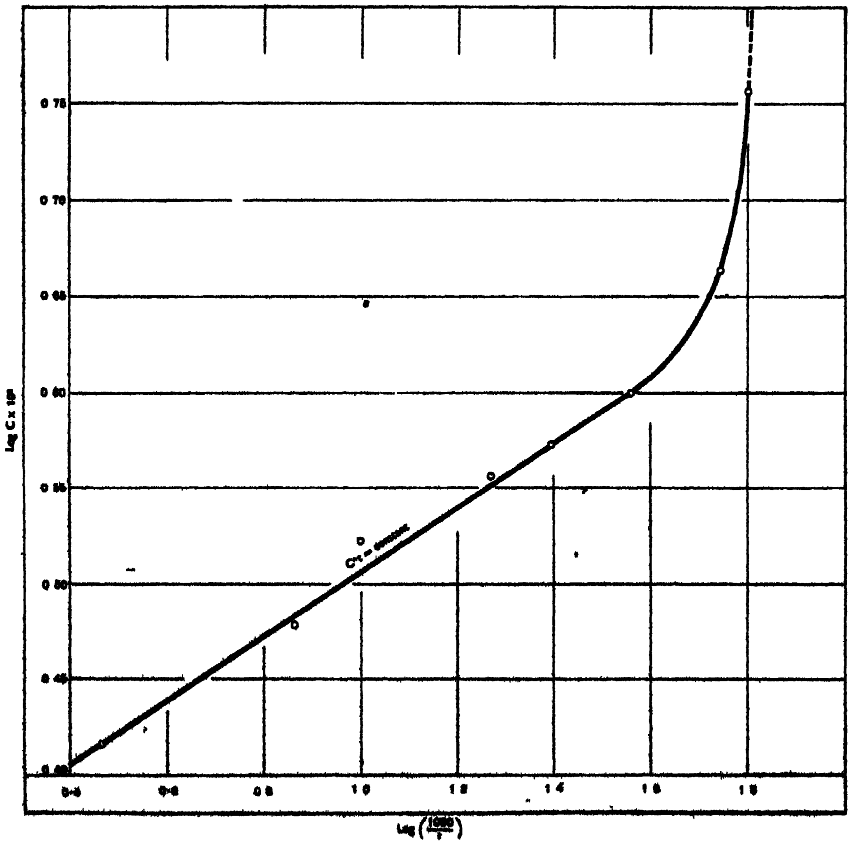


Fig. 6.

ACTION UPON THE ISOLATED INTESTINAL STRIP.

Strips taken from the small intestine of rabbits or sheep were suspended in Tyrode solution in the Dale bath and contractions kymographically recorded. The addition of Icterogenin A, B or C, as the neutral sodium salt dissolved in a small quantity of distilled water, was found to paralyse the tissue at concentrations as low as 1 in 200,000. The inhibition was permanent at higher concentrations but by removal of the bathing fluid and substitution of fresh Tyrode solution, intestinal strips poisoned by 1/200,000 icterogenin slowly recovered their normal rhythm. Repetition of the dose again caused inhibition. The muscle in every case remained fully *relaxed*. It is thus possible to visualise the manner in which the *Lippia* toxic principle brings about such intense constipation and ruminal inhibition in the living animal. See Figs. 9, 10, 11 and 12.

BLOOD PRESSURE AND PULSE RATE.

Injection of 0·1 gm. Icterogenin intravenously into the anaesthetised animal (dog) causes only a slight diminution in pulse rate accompanied by an increase in the pulse interval. There is no effect upon the general systolic pressure. See Fig. 13.

PERFUSION OF THE ISOLATED HEART.

Rabbits' hearts were perfused with Ringer-Locke solution and neutral solutions of the sodium salt of Icterogenin added to the perfusion fluid. Quantities of 1 mgm, of Icterogenin caused a pronounced slowing of the heart beat and decrease in amplitude. The onset was gradual but the effect increased until the heart was arrested in systole. With larger quantities (1·5 mgm.) an almost immediate inhibition occurred, the muscle remaining fatally contracted in a systolic spasm —see Figs. 14 to 16. The exact concentration is difficult to give since the Icterogenin solution was added to the moving column of liquid.

DISTRIBUTION OF ICTEROGENIN BETWEEN LEAVES, STEMS AND ROOTS OF *Lippia* PLANTS THROUGHOUT THE YEAR.

When examining a batch of *Lippia* plants growing in the poison garden at Onderstepoort, the observation was made that the roots of the plant possessed a thick, fleshy bark and by trial, crude Icterogenin was found to be present in this tissue in comparatively high concentration. Analysis of the different parts of the plant, using the sodium salt technique and working with care to ensure quantitative isolation, afforded the following figures in terms of sodium salt per 100 grams of air-dried material.

Onderstepoort poison garden *Lippia*, collected 6/4/36.

Leaves: 1·3 kilos yielded 1·251 gm. — 0·096 per cent.
Stems: 400 gms. yielded a trace — trace only.
Whole roots: 350 gms. yielded 2·5 gm. — 0·71 per cent.
Root wood: 120 gms. yielded a trace — trace only.
Root bark: 420 gms. yielded 8·556 gms. — 2·04 per cent.

It is clear from the above figures that the greatest concentration of this toxic material is to be found in the root bark, that of the leaves being in the particular sample investigated some 20 times

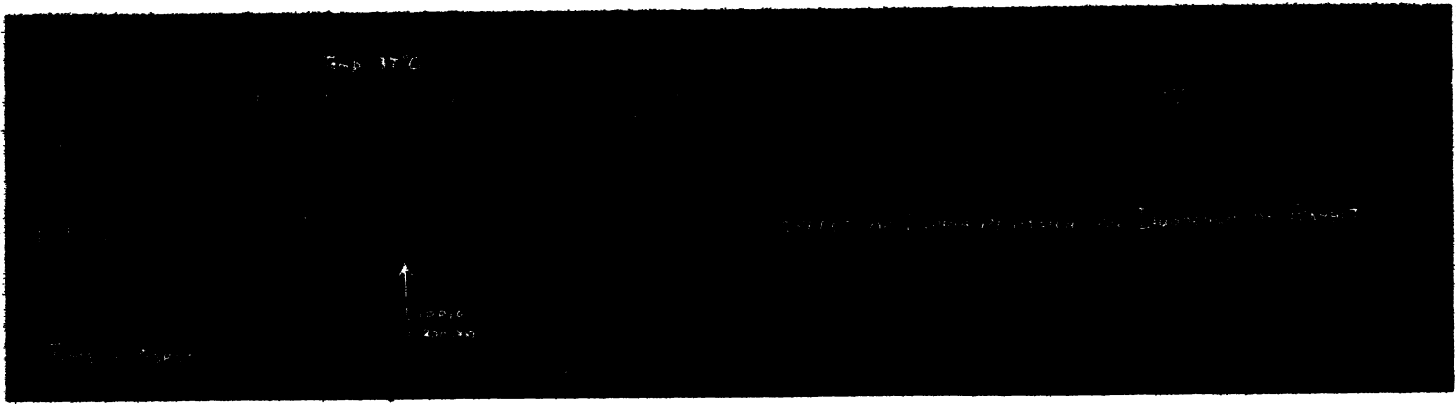
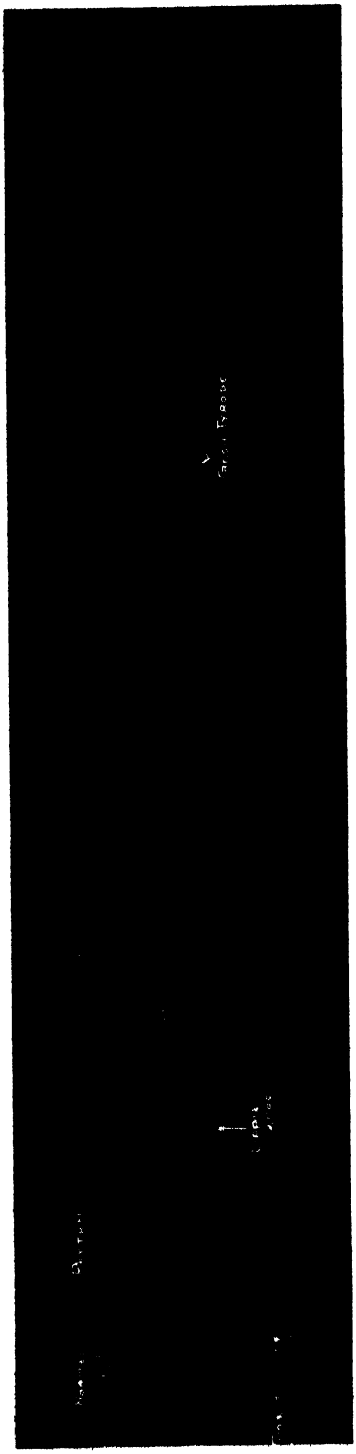


Fig. 9. Effect of Icterogenin 1/200.000 on rabbit duodenal strip.



Interval 146 seconds.

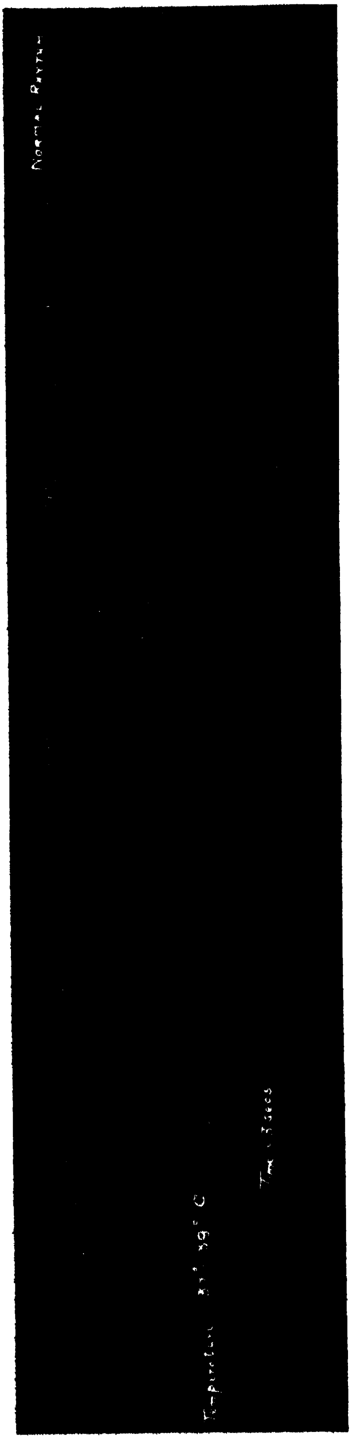


Fig. 10. Effect of Icterogenin 1/100,000 on rabbit duodenal strip showing inhibition and recovery.

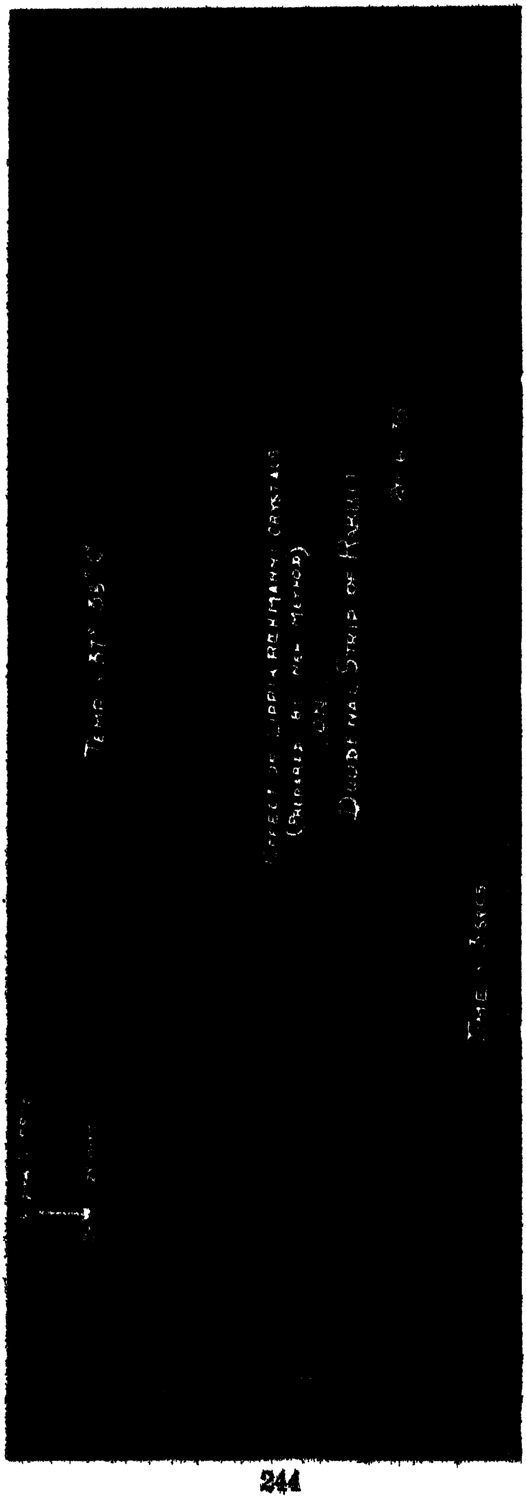


Fig. 11. Effect of Icterogenin B 1/25.000 (prepared by sodium salt method) on rabbit duodenal strip.

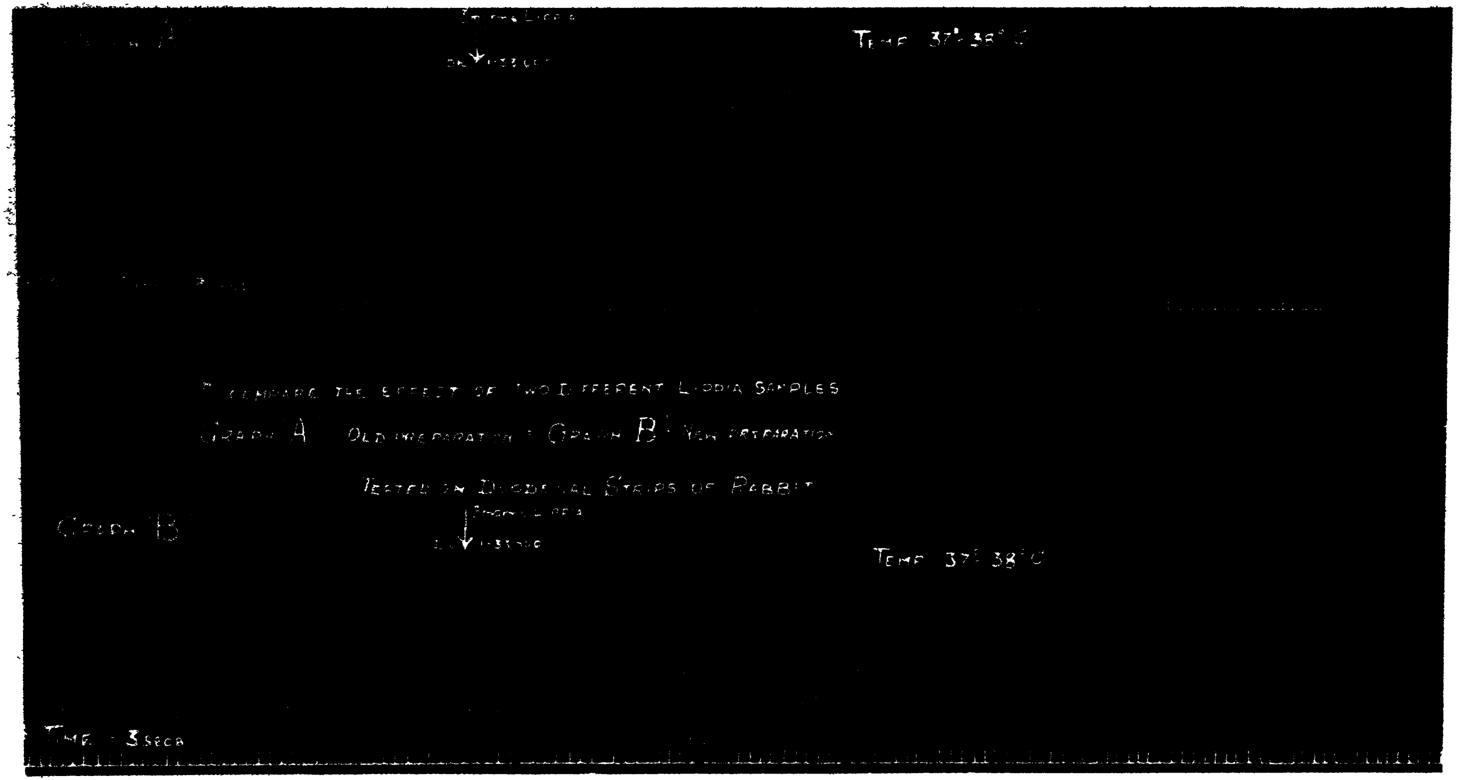
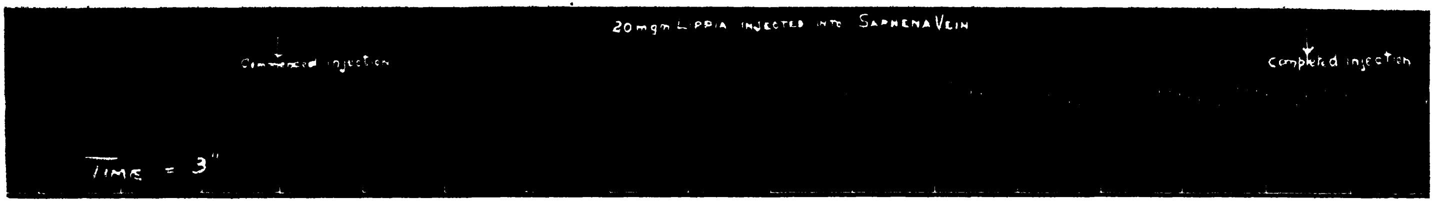


Fig. 12. Comparison of effects of Icterogenins A and B on rabbit duodenal strip.



Interval 195 seconds.

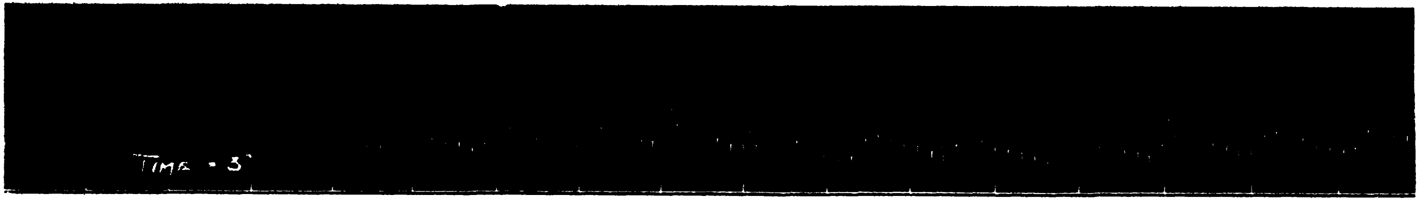


Fig. 13. Intravenous injection 0·1 gm. Icterogenin into a dog: effect on pulse rate and pressure.

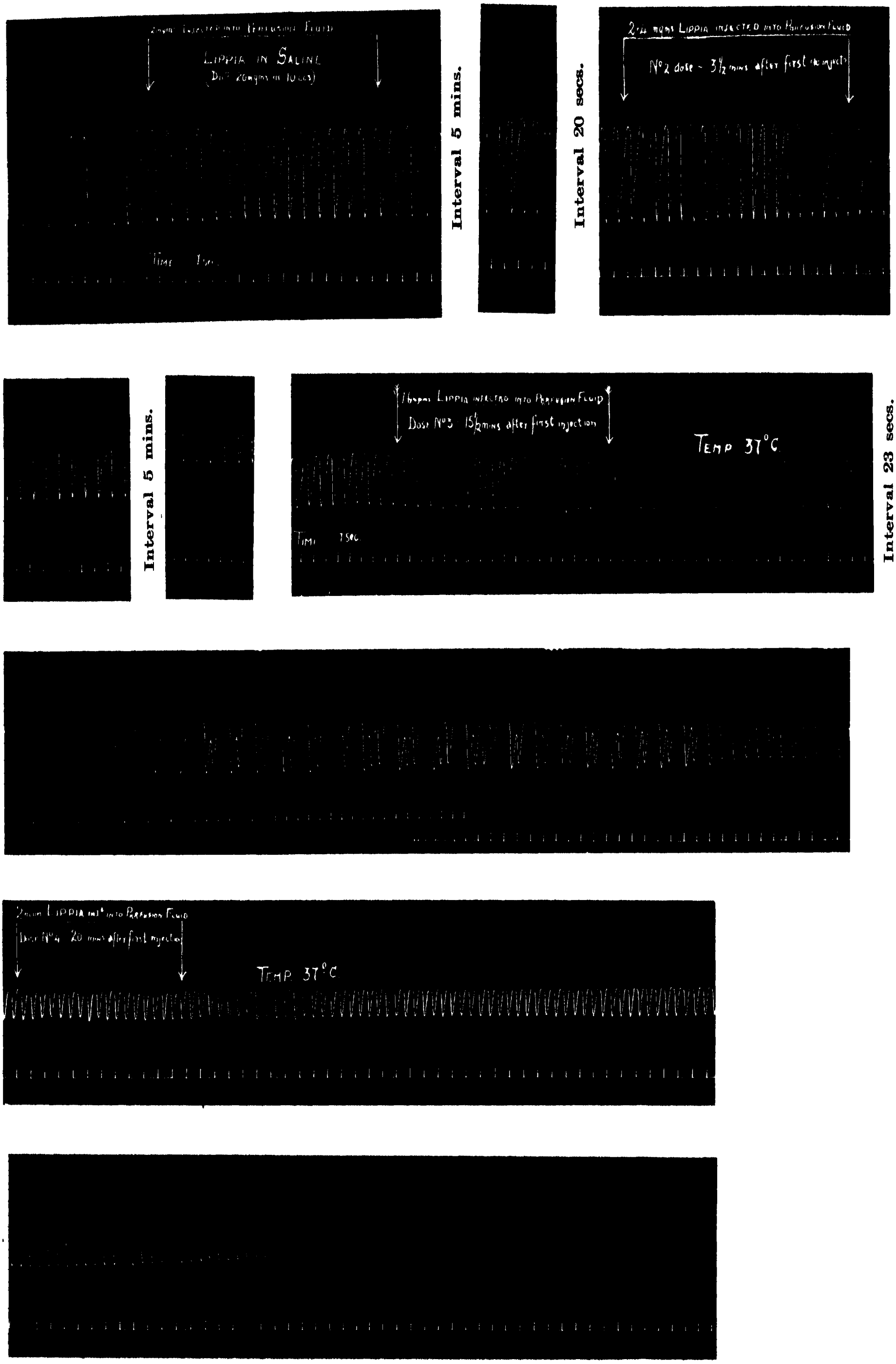


Fig. 14.—Effect of Icterogenin upon the isolated, perfused rabbit's heart.

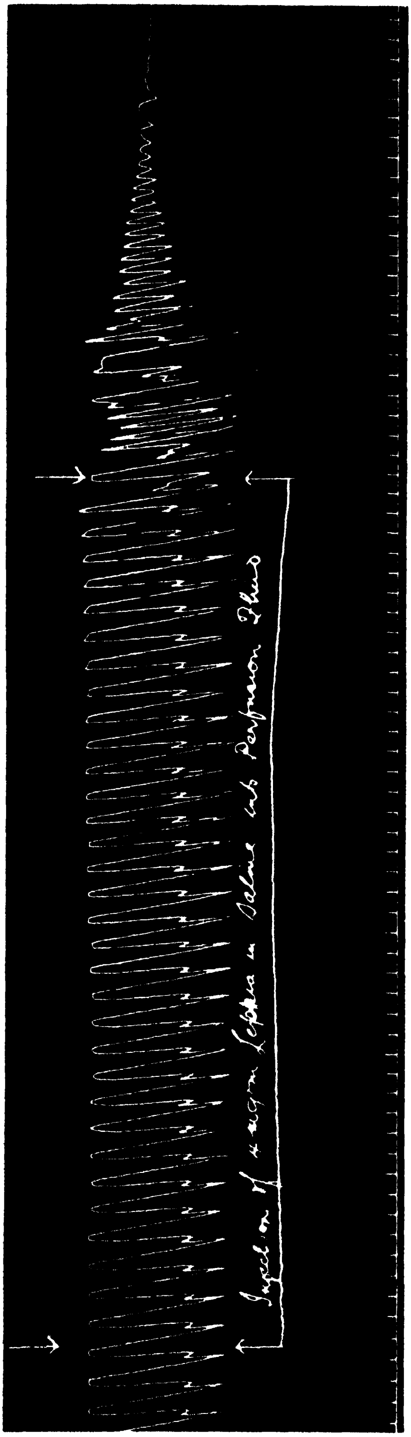


Fig. 15.—Effect of Icterogenin (4 mgm.) upon the isolated, perfused rabbit's heart. Time interval 1 sec.; temp. 35·7°.

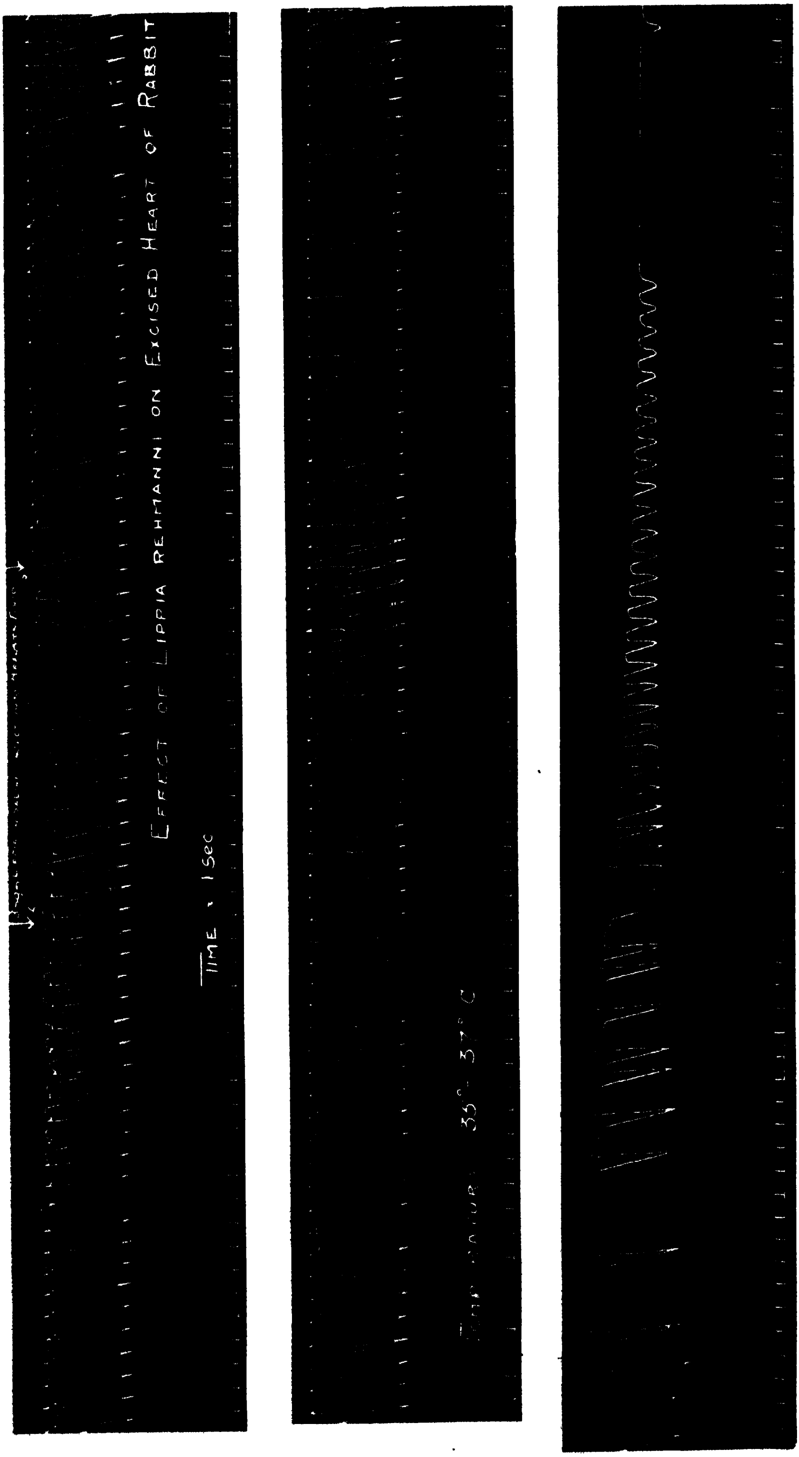


Fig. 16.—Effect of Icterogenin (3 mgm.) upon the isolated, perfused rabbit's heart.

less, also that the woody portions both of stem and roots are virtually free from Icterogenin. It may be remarked that these plants were in the post-seeding stage when gathered and that heavy summer rains had fallen not long before.

Since this finding promised to throw considerable light on the fluctuations in toxicity throughout the year both of *Lippia* and possibly also of *Tribulus*, the cause of geel-dikkop, the following experiment was designed.

TABLE VI.

Distribution of Icterogenin in Lippia *plants during the year.*

Month.	Date of Collection.	Leaves gm/100 gm. air dry weight.	Root bark gm/100 gm. air dry weight.	State of growth.	Rainfall in inches.
1936.					
Mar....	—	—	—	—	8·50
April...	—	—	—	—	0·51
May....	6th duplicate	0·17 0·16	1·58 1·59	Very dry; seeds falling out	4·26
June...	—	—	—	—	Nil.
July....	—	—	—	—	Nil.
Aug....	—	—	—	—	Nil.
Sept....	28th	0·28	1·01	Young, green shoots but ground very dry	0·46
Oct....	—	—	—	—	2·68
Nov....	5th	0·12	1·76	Plants fresh after good rains..	6·53
Dec....	10th	0·10	3·38	Period of drought prior to collection. Root bark shows spongy new growth	3·37
1937.					
Jan.....	—	—	—	—	4·51
Feb....	—	—	—	—	10·64
Mar....	11th	0·32	2·54	Bushes in good condition after heavy summer rains	1·38
April...	27th 27th	0·26 0·96	1·92 4·40	(Unpruned bushes)............ (Leaves and root bark from bushes pruned March 11th, see text).	2·20

These results are recorded graphically in Fig. 17.

A plot of natural, ungrazed veld, heavily overgrown with *Lippia rehmanni*, was kindly placed at our disposal by Mr. J. Wolmarans of the farm "Derdepoort," Silverton, Pretoria district. We wish to express our thanks to Mr. Wolmarans for his generous collaboration. At intervals during the year, expeditions were made to the farm and a sufficient quantity of *Lippia* plants uprooted to allow of chemical investigation. The roots were washed under a tap to remove adhering soil and the fleshy root bark then removed and spread out to dry: the leaves were easily plucked from the stems after sun-drying. Stems were not as a rule investigated after a preliminary experiment had shown that traces only of Icterogenin could be isolated from them. Each material was finely ground and then extracted according to the technique described in this paper. It was found that the root bark could be directly extracted by boiling ether in a Soxhlet apparatus thereby shortening the procedure by one stage. The yields of sodium salt were recorded and it was found that duplicate analyses agreed well. In the above table (Table VI), the results are reproduced together with the monthly rain-fall figures measured at Onderstepoort.

An experiment was made on March 11th whereby it was hoped to ascertain if new growth was always to be associated with high Icterogenin content, and also whether or not Icterogenin was transferred from the leaves to the root bark. A number of bushes was stripped of leaves and pruned back very severely. At the date of the next collection, a month later, we were gratified to observe that this had had the desired effect of stimulating the plants to shoot. A mass of tender green leaves covered the old stems. These leaves were gathered, the plants then dug up and the bark from the roots also collected separately for comparison with the corresponding tissues of the untreated bushes. It was found that not only was the Icterogenin content of the leaves the highest which we have ever recorded (0·96 per cent.) but that the quantity in the root bark had also risen markedly. In the untreated plants, a fall was recorded in both leaves and bark.

We feel quite justified in drawing the conclusions therefore that (*a*) a transference of Icterogenin from leaves to root bark does normally occur and (*b*) that the growth of young leaves is associated with a big production of Icterogenin. It seems doubtful if any synthesis of icterogenin could take place in the root bark *in situ* so that the rise and fall occuring in this tissue during the course of the year must be occasioned by the transference of material to or from the leaves. The bark, of course, exhibits growth at certain seasons of the year, thus in December (mid-summer) the whole tissue was soft, fleshy and but poorly suberised, but since all quantities are calculated upon the dry weight basis, as a percentage, the resultant figure will embrace both of the two factors—the quantity of Icterogenin passed to the roots and the bulk of the bark itself.

Examination of the graph, Fig. 17, shows that the transference from leaves to root must be fairly rapid, in the early spring at least. About November, a fall in leaf Icterogenin is reflected by a rise in that of the root bark, as indicated by the crossing of the curves; later on (April) both values fall together. The precise influence of rainfall, apart from growth, if any such influence exists, will only become evident when we have a larger number of values extending over several seasons.

Whether or not a transference of Icterogenin in the reverse direction may take place, that is from root to the above ground portions of the plant, is a question we would like to leave open. We realise that hypothetical transference in this direction does not accord with generally accepted views, nevertheless, we do not wish to loose sight of the possibility.

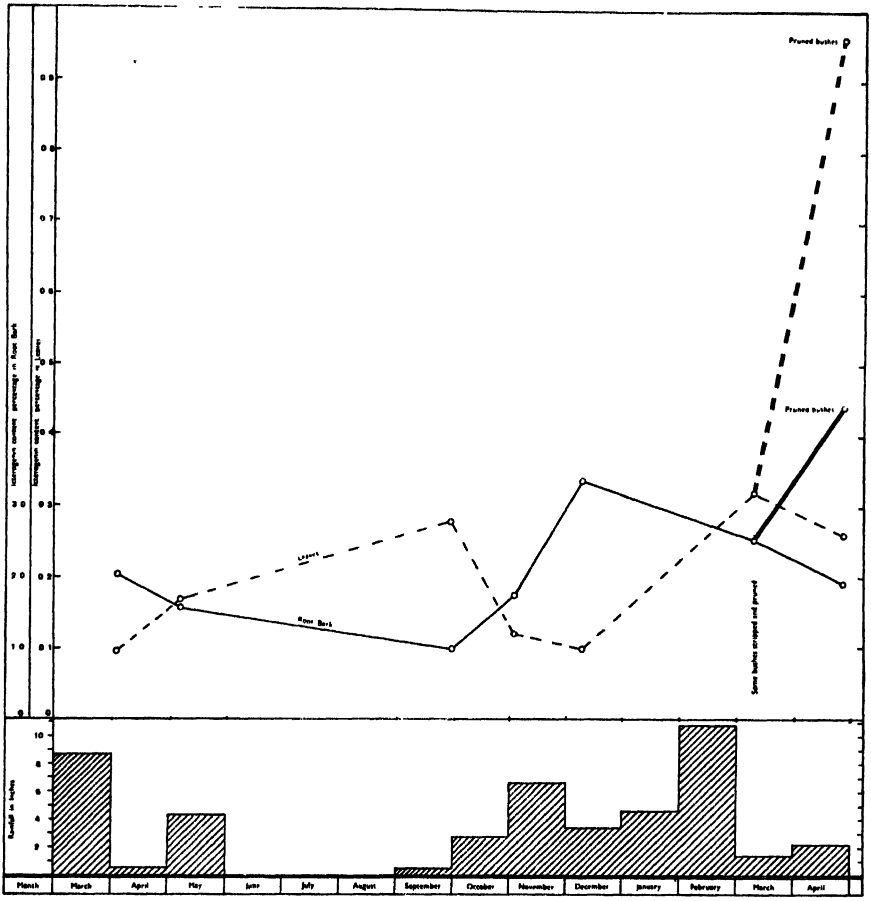


Fig. 17.

Finally, we would like to indicate briefly the bearing that these experiments may have upon the seasonal nature of geel-dikkop outbreaks and the well known tendency for this disease to disappear after good rains. Admittedly, many factors must contribute to the capricious nature of geel-dikkop, among which must be reckoned the scarcity or otherwise of other foodstuffs, the condition of the sheep, etc., but farming experience has shown that light rains leading to rapid growth of *Tribulus*, followed by a period of hot, dry weather, are indicative of danger from geel-dikkop. Such conditions, following the *Lippia* model, would be conducive to a rapid rise in the Icterogenin content of the leaves of this plant. December to March is also the period at which the danger from geel-dikkop is at its greatest.

Experiments are still in progress designed to reveal seasonal fluctuations in the toxicity of *Tribulus* and the root system of this plant is also now receiving due attention.

SUMMARY.

1. An improved method is described for the isolation of the icterogenic material from *Lippia rehmanni* Pears. (Verbenaceae). This takes advantage of the sparing solubility of the sodium salt of the active material in solutions containing sodium ions.

2. In addition to the two acids previously described, namely prisms M.P. 239° and irregular plates melting with loss of weight at 158°, resolidifying to melt ultimately at about 230°, a third active acid has been found to be present in the mixture of crude sodium salts. This material crystallises in elongated regular plates, M.P. 161° without resolidification or loss in weight. It is proposed to designate these materials Icterogenin A, C and B respectively.

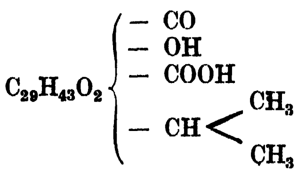
3. Comparison of the analytical data afforded by the free acids and their acetyl derivatives with microtitration experiments and the nitrogen content of the 2:4 dinitrophenylhydrazones, leads to the formula $C_{34}H_{52}O_6$ as most probably representing the true composition of all three isomers. The possibility of further substances being present in the mixture is not excluded.

4. In alcoholic solution, the Icterogenins A, B and C have optical rotatory powers of +72·0°, +69·0° and +70·5° respectively.

5. Acetyl derivatives of Icterogenins A and B were prepared and found to be identical. The substance crystallised in needles M.P. 142°. Saponification showed that one hydroxyl group had been acetylated.

6. The presence of one ketonic group in the molecule was shown by the preparation of the 2:4 dinitrophenylhydrazones crystallising in orange-yellow needles with M.P.'s 222-5°, 226-9° and 224-8° respectively.

7. Chromic acid oxidation indicated the presence of an iso-propyl side chain. The formula of Icterogenin can thus be written



$$\mathrm{C_{29}H_{43}O_2}\left\{\begin{array}{l} -\mathrm{CO} \\ -\mathrm{OH} \\ -\mathrm{COOH} \\ -\mathrm{CH}<\begin{array}{l}\mathrm{CH_3}\\ \mathrm{CH_3}\end{array}\end{array}\right.$$

8. Icterogenin, in a dose of 1·5 gm. or more *per os* to a sheep, causes bilirubinaemia within 24 hours, together with atony and stasis in the fore stomachs and large intestine.

9. By the intravenous route it was very toxic, quantities of 1 gm. causing death with shock-like symptoms. Lesser amounts cause bilirubinaemia and hurried respiration.

10. Upon the general systolic blood pressure, Icterogenin has no effect in doses of 0·1 gm. to a dog, but the pulse is slightly retarded and the pulse interval appreciably increased.

11. On the isolated heart, Icterogenin has a pronounced inhibitory effect in quantities of 1 mgm. added to the perfusion fluid. A systolic spasm gradually develops which, with larger doses, proves fatal.

12. The icterogenins inhibit the isolated intestinal strip in a concentration of 1 in 200,000, the muscle becoming relaxed.

13. Icterogen is haemolytic *in vitro* in a concentration of 1 in 35,000.

14. Examination of the root bark of *Lippia rehmanni* shows that the highest concentration of Icterogenin is present in this tissue.

15. A quantitative experiment extending over a year shows that a translocation of Icterogenin takes place from the leaves to the root bark in *Lippia* plants. The effect of growth, season, etc. upon this mechanism has been studied and the bearing of the results upon the incidence of geeldikkop is discussed.

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Section X.

Sheep and Wool.

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Wool Studies.

II. The Frequency Distribution of Merino Wool Fibre Thickness Measurements.

By A. P. MALAN, Section of Statistics, Onderstepoort.

1. INTRODUCTION.

Practically the whole of the statistical theory and practice of modern agricultural and biological experimentation is based on what is known as the *normal theory*. This means that the usual tests of significance of statistical coefficients are based on the assumption that the coefficients are estimated from " random samples from a normal population ".

Although the existing tables for significance tests are mostly based on the assumption of a normal parent population, it is known that the applicability of these tests is not always confined to strictly normal populations. It was demonstrated with a practical example by Eden and Yates (1933) that the lack of normality did not violate the application of Fisher's z-tests in the analyses of variance. However, it is equally true that the normal theory may not be applied indiscriminately to all data. It is therefore necessary to study the nature of the observed distribution functions in different fields of experimental work.

In cases where the observed variate is definitely not normally distributed it is sometimes possible to substitute a function of the observed variate as the new variable quantity, which becomes normally distributed. So, for instance in cases where the observed standard deviations for different samples vary in proportion to the respective mean values, it is reasonable to use the logarithms of the observed values as the variate for the statistical analysis of the data.

2. WOOL FIBRE THICKNESS MEASUREMENTS.

Fibre thickness is measured at the Onderstepoort Wool Laboratory by the micro-camera method. The sampling consists of zoning the original sample and from each of these a small portion is taken and combined into a single sample. This sample is cut along its whole length into a large number of small fragments which are then mixed in a beaker of ether. A portion of this mixture is taken at random, dried and mounted in Euparal on a slide from which the required number of thicknesses is determined by means of a Zeiss-Hegener micro-camera.

The above method allows for the variation in thickness between fibres and the variation in thickness along the length of the same fibre, but in an uncontrolled way, in the sense that some fibres may contribute more to the observed variation than others according to the respective numbers of fragments included in the observed values. Various other objections may be made against the above procedure, and the involved problem of wool sampling is receiving a thorough investigation. It is hoped to give a more detailed discussion of wool sampling technique in a future study.

Fibre thickness measurements are known to have a skew frequency distribution which is not normal. The actual nature of this distribution has, to the author's knowledge, never been discussed and it is the intention of this paper to apply the logarithmic transformation to observed thickness measurements. This transformation was suggested by the constancy of the coefficients of variability for the same fibre population.

In Study I, Malan, van Wyk and Botha (1935) considered amongst others, the variation in fibre thickness measurements over a period of three years. Consecutive measurements were made of shoulder samples from a marked area on the skin to ensure that they represented the same fibre population. A striking feature of the results was the constancy of the coefficients of variability obtained for different years, notwithstanding a considerable change in the mean values. In a paper on some characteristics which enter into the assessment of wool quality, and their estimation in the fleece, Wildman (1935) used the logarithms of fibre thickness " on account of the proportionate relation between the standard deviation and mean ". This transformation into logarithms would be justified if the logarithms become normally distributed.

3. THE LOGARITHMIC DISTRIBUTION:

A variate x is said to be normally distributed when the frequency in an infinitesimal interval dx is proportional to df, where:

$$df = \frac{1}{s\sqrt{2\pi}}\, e^{-\frac{(x-a)^2}{2s^2}}\, dx \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (1)$$

Hence if the variate (x) in (1) is considered as the natural logarithm of fibre thickness (t), $x = log_e\,(t)$, the distribution function of t is given by:

$$df = \frac{1}{\sigma\sqrt{2\pi}}\, e^{-\frac{1}{2\sigma^2}\log_e^2\left(\frac{t}{m}\right)}\, \frac{dt}{t} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (2)$$

In (2) the parameters are m and σ, where m is the geometrical mean of the fibre thickness measurements and σ is the root mean square deviation of $log_e\, t$ from the natural logarithm of the geometrical mean. Evidently, therefore, σ is a measure of mean squared deviation as a proportion of the mean and $100\,\sigma$ is a measure of compound percentage deviation from the mean. The coefficient, σ, will be referred to as the *coefficient of " relative " variability.*

The properties of the above function (2) have been considered by various authors but the required results for its application in this paper are again deduced and graphically illustrated.

This function, (2), has its maximum where:

$$t = m\, e^{-\sigma^2} \quad \text{.......................................}(3)$$

and its points of inflexion at:

$$t = m\, e^{-\frac{3}{2}\sigma^2 \pm \sigma\sqrt{1+\frac{\sigma^2}{4}}}$$

The moments (M') about the origin, where t is *zero*, are given by:

$$M'_n = \frac{1}{\sigma\sqrt{2\pi}} \int_0^\infty t^n\, e^{-\frac{1}{2\sigma^2}\, log_e^2\left(\frac{t}{m}\right)}\, \frac{dt}{t}$$

, which by putting

$\frac{1}{\sigma\sqrt{2\pi}}\, log_e\left(\frac{t}{m}\right) = x$, is easily shown to give:

$$M'_n = m^n\, e^{\frac{n^2\sigma^2}{2}} \quad \text{.......................................}(4)$$

From (4) the first two moments are obtained by putting $n = 1$ and 2 respectively:

$$M'_1 = m\, e^{\frac{\sigma^2}{2}} = a, \text{ the arithmetical mean } \text{..........................}(5)$$

$$M'_2 = m^2\, e^{2\sigma^2} \quad \text{.......................................}(6)$$

Hence the parameters m and σ, expressed in terms of the first two moments are:

$$m = \frac{M_1'^2}{\sqrt{M'_2}} \quad \text{.......................................}(7)$$

$$e^{\sigma^2} = \frac{M_2'}{M_1'^2} \quad \text{or} \quad \sigma^2 = log_e\left(\frac{M_2'}{M_1'^2}\right) \quad \text{..........................}(8)$$

From the above moments about zero (M') given by (4), the moments about the arithmetical mean (M), are found to be:

$$M_n = m^n\, e^{\frac{1}{2}n\sigma^2} \sum_{i=1}^{n} \left\{ (-1)^i \binom{n}{i}\, e^{\frac{1}{2}(n-i)(n-i-1)\sigma^2} \right\} \text{......}(9)$$

Likewise, having got the moments, the cumulants or semi-invariants may be obtained by means of the known relationships between them.

From (9) the first two moments about the arithmetical mean, $a = M'_1$ are:

$M_1 = 0$

$M_2 = m^2 e^{\sigma^2}(e^{\sigma^2} - 1) = s^2$ ....(10)

where s^2 is the ordinary variance.

Substituting from (5), which gives the arithmetical mean, a, the above equation (10) becomes:—

$s^2 = a^2(e^{\sigma^2} - 1)$ ....(11)

$$\frac{s^2}{a^2} = e^{\sigma^2} - 1$$

$$= \sigma^2 + \frac{\sigma^4}{2!} + \frac{\sigma^6}{3!} + - - - -$$

$\bumpeq \sigma^2$ to a first approximation, and $\bumpeq \sigma^2(1 + \frac{\sigma^2}{4})^2$ to a second approximation.

From (11) the value of σ may be expressed in terms of s/a as follows:

$\sigma^2 = \log_e\left(1 + \frac{s^2}{a^2}\right)$ ....(12)

$$= \frac{s^2}{a^2} - \frac{1}{2}\frac{s^4}{a^4} + \frac{1}{3}\frac{s^6}{a^6} - - - -$$

$= \frac{s^2}{a^2}$ to a first approximation, and

$= \frac{s^2}{a^2}\left(1 - \frac{1}{4}\frac{s^2}{a^2}\right)^2$ to a second approximation.

The following table illustrates the accuracy of the second approximation for σ in terms of s/a where 100σ was called the *coefficient of relative variability* and $100\,s/a$ the ordinary *coefficient of variability.*

TABLE 1.

s/a.	0·1	0·2	0·3	0·4	0·5
$\sigma = \sqrt{\log_e\left(1 + \frac{s^2}{a^2}\right)}$	0·099,751	0·198,040	0·293,560	0·385,253	0·472,380
$\frac{s}{a}\left(1 - \frac{1}{4}\frac{s^2}{a^2}\right)$	0·099,750	0·198,000	0·293,250	0·384,000	0·468,750
Difference..............	0·000,001	0·000,040	0·000,310	0·001,253	0·003,630

From the differences in the last row it is clear that for a coefficient of variability below 30 per cent. the second approximation for σ is sufficiently accurate for most purposes.

4. THE FITTING OF THE LOGARITHMIC CURVE.

The statistical coefficients m and σ for any observed distribution may be estimated from the first two moments about zero by means of the relations (4) and (5) respectively. Once the estimates of m and σ are known the fitting of the theoretical curve (2) to the observed frequencies, is a matter of routine procedure. The expected frequency of values between *zero* and t is proportional to:

$$A_t = \frac{1}{\sigma\sqrt{2\pi}} \int_o^t e^{-\frac{1}{2\sigma^2}\log_e^2\left(\frac{t}{m}\right)} \frac{dt}{t} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots(13)$$

By putting $\frac{1}{\sigma}\log_e\left(\frac{t}{m}\right) = x$ the above integral takes the familiar form,

$$A_t = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{x} e^{-\frac{1}{2}x^2}\, dx \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots(14)$$

Therefore the area of the "tail" of the logarithmic curve from o to t is equal to the area of the "tail" of the normal error function from $-\infty$ to $m\,e^{\sigma x}$. Hence by putting $x = \frac{1}{\sigma}\log_e\left(\frac{t}{m}\right)$ the required values of x for the given function to enter, e.g. Table II of Pearsons' *Tables for Statisticians and Biometricians, Pt. I*, are obtained.

Thus, when the total observed frequency is equal to N, the "expected" frequency between t_1 and t_2 is given by:

$$A_{t_2} - A_{t_1} = \frac{N}{\sigma\sqrt{2\pi}}\left[\int^{t_2} e^{-\frac{1}{2\sigma^2}\log_e^2\left(\frac{t}{m}\right)} \frac{dt}{t} - \int^{t_1} e^{\frac{1}{2\sigma^2}\log_e^2\left(\frac{t}{m}\right)} \frac{dt}{t}\right]$$

$$= \frac{N}{\sqrt{2\pi}}\left[\int_{-\infty}^{x_2} e^{-\frac{x^2}{2}}\,dx - \int_{-\infty}^{x_1} e^{-\frac{x^2}{2}}\,dx\right]$$

Where $x_1 = \frac{1}{\sigma}\log_e\left(\frac{t_1}{m}\right)$ and $x_2 = \frac{1}{\sigma}\log_e\left(\frac{t_2}{m}\right)$

By calculating the "expected" frequencies for each group interval, the agreement between "observed" and "expected" frequencies may be tested by means of the χ^2 test for "goodness of fit".

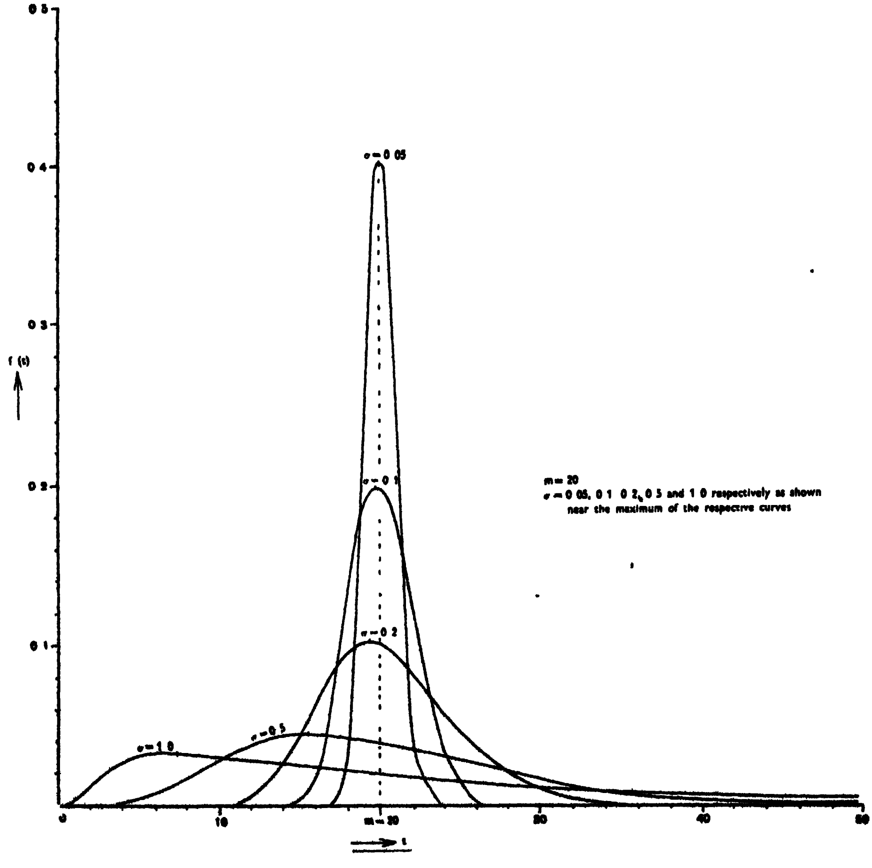
To draw the theoretical curve, estimated from the observed data, it is only necessary to note that the ordinate at any point t ($m\,e^{\sigma x}$) is obtained by multiplying the error function value $\left(\frac{1}{\sqrt{2\pi}}e^{-\frac{1}{2}x^2}\right)$ for $x\left(= \frac{1}{\sigma}\log_e\left(\frac{t}{m}\right)\right)$ by $\frac{N}{\sigma t}$.

5. THE SHAPE OF THE LOGARITHMIC CURVE.

The normal function (1) has a familiar bell shaped form and is symmetrical about the mean (a), where the function has its maximum value. The variate x varies from $-\infty$ to $+\infty$ and the shape of the curve is flattened out with increasing values of the standard deviation s.

By the logarithmic transformation the function (2) becomes skew and the variate (t) remains positive between the limits $o \leq t \leq \infty$, with its maximum at the point $t = m\, e - \sigma^2$ as given by (3). For the same value of the geometrical mean, the maximum point moves further away from the mean towards *zero* as σ, the *coefficient of relative deviation*, increases. The alteration in the shape of the logarithmic curve (2) for different values of σ is illustrated by *Chart A*, where the logarithmic curves are drawn with a constant geometrical mean $m = 20$ and *coefficients of relative deviation*, $\sigma = 0{\cdot}05$, $0{\cdot}1$, $0{\cdot}2$, $0{\cdot}5$ and $1{\cdot}0$ respectively. For these curves the maximum values are at $t = 19{\cdot}95$, $19{\cdot}80$, $19{\cdot}22$, $15{\cdot}58$ and $7{\cdot}36$ respectively. The lack of normality is clearly increased by increased values of σ,

CHART A.—The Logarithmic Function $f(t) = \frac{1}{\sigma\sqrt{2\pi}}\, e^{-\frac{1}{2\sigma^2} \log_e^2 \frac{t}{m}}\, \frac{1}{t}$



6. THE PARAMETERS OF THE NORMAL AND LOGARITHMIC FUNCTIONS.

The normal function (1) is uniquely determined by the parameters *a* and *s* while the corresponding logarithmic function is determined by *m* and σ. The relationships between these parameters have been deduced and given in paragraph 3. It is of interest to consider the difference between these parameters when the two functions are applied to the same population. For this purpose populations have been selected with a constant arithmetical mean, (*a*), equal to 20 and standard deviations varying from 1 to 40, i.e. coefficients of variability from 5 per cent. to 200 per cent. These extreme values for the coefficients of variability are included because the shapes of the corresponding logarithmic curves depends only, according to (12), on the ratio of the standard deviation to the mean. The mean of 20 has been taken to represent more or less an average value for fibre thickness measurements. Obviously, when any other mean (*a*) is chosen with the same coefficient of variability the logarithmic deviation coefficients remain unaltered and specific values for the variates are obtained from the results given below by simply multiplying the given values by the ratio $^{m}/20$.

The position is clearly illustrated by Table I where the logarithmic values of the mean, maximum point and deviation coefficients are given for each coefficient of variability. This table also includes columns to show the differences between the means and between deviation coefficients. The increased skewness of the logarithmic curve for greater values of the deviation coefficients is further illustrated by the difference column between the geometrical mean (*m*) and the maximum point (*T max*).

TABLE II.

a – 20. *s*	*s*/*a*	*m*	T *max*	σ	*a*—*m*	*m* — T *max*	100 x (*s*/*a*—σ)
1	0·05	19·975	19·925	0·04997	0·025	0·025	0·003
2	0·10	19·901	19·704	0·09975	0·099	0·197	0·025
3	0·15	19·779	19·343	0·14917	0·221	0·436	0·083
4	0·20	19·612	18·857	0·19804	0·388	0·755	0·196
5	0·25	19·403	18·262	0·24622	0·597	1·141	0·378
6	0·30	19·157	17·575	0·29354	0·843	1·582	0·646
8	0·40	18·570	16·008	0·38525	1·430	2·562	1·475
10	0·50	17·880	14·311	0·47234	2·111	3·578	2·766
12	0·60	17·150	12·610	0·55451	2·850	4·540	4·549
15	0·75	16·000	10·240	0·66805	4·000	5·760	8·195
20	1·0	14·144	7·072	0·83255	5·856	7·072	16·745
24	1·2	12·804	5·247	0·94446	7·196	7·557	25·554
30	1·5	11·094	3·414	1·0850	8·906	7·680	41·50
36	1·8	9·713	2·291	1·2019	10·287	7·422	59·81
40	2·0	8·944	1·789	1·2686	11·056	7·155	73·14

a = arithmetical mean,
s = normal standard deviation,
m = geometrical mean,
σ = logarithmic or ‘ relative ’ deviation coefficient,
T *max* = point where the logarithmic curve has its maximum.

7. The Probability Integral when the Parameters of the Normal Population are used for a Logarithmic Distribution.

For the Normal Population given by (1), the area under the curve beyond a point $x = a + ns$ is given by $1 - P_{ns}$ where:

$$P_{ns} = \frac{1}{s\sqrt{2\pi}} \int_{-\infty}^{a+ns} e^{-\frac{(x-a)^2}{2s^2}}\, dx \qquad \text{.........(15)}$$

The total area outside the limits $a \pm ns$ is given by $2\ (1 - P_{ns})$, which is equal to:—

$$\left\{ 1 - \frac{1}{s\sqrt{2\pi}} \int_{a-ns}^{a+ns} e^{-\frac{(x-a)^2}{2s^2}}\, dx \right\} \qquad \text{.........(16)}$$

Using the limits $t = a \pm ns$ for the logarithmic population (2) the integral (15) becomes:

$$\frac{1}{\sigma\sqrt{2\pi}} \int_{-\infty}^{a+ns} e^{-\frac{1}{2\sigma^2}\log_e^2\left(\frac{t}{m}\right)}\, \frac{dt}{t} \qquad \text{.........(17)}$$

where m and σ are given by (7) and (8), and the value of (16) is given by:

$$1 - \frac{1}{\sigma\sqrt{2\pi}} \int_{a-ns}^{a+ns} e^{-\frac{1}{2\sigma^2}\log_e^2\left(\frac{t}{m}\right)}\, \frac{dt}{t} \qquad \text{.........(18)}$$

Hence, to obtain the value of these integrals, it follows from paragraph (4) that the values of these limits by which Pearson's *Tables for Statisticians and Biometricians* is to be entered, are:

$$t = \frac{1}{\sigma} \log_e \left(\frac{a \pm ns}{m} \right)$$

These limits are affected by the arithmetical mean and the standard deviation, or to be more accurate, by the ordinary coefficient of variability. It has, therefore, been decided to consider all the values of $^v/_a$ from 0·05 to 2. The probabilities of obtaining values of t below $(a - ns)$, beyond $(a + ns)$ and both below and beyond these two values of t, where $n = 1$, 2 and 3 and the distribution of t are given by the logarithmic function (2). These probabilities for $n = 1$, 2 and 3 are represented by charts B, C and D respectively. The probabilities of getting values of $t \leq a - ns$ and $t \geq a + ns$, written $P\{t \leq (a - ns)\} = P_1$ $P\{t \geq (a + ns)\} = P_2$, and also the values for $P_1 + P_2$, as $^v/_a$ varies from 0 to 2, are shown by curves on these charts. Furthermore the total probabilities (P) of getting deviations from the mean greater than *one, two* or *three* times the standard deviation, σ, are shown by dotted lines on the respective charts. Hence the difference between the dotted line value and the value on the $P_1 + P_2$ line for a particular value of $^v/_a$ show the difference between the assumed normal theory value and the actual value based on the logarithmic distribution.

CHART B.—Giving $P\{t < (a-s)\} = P_1$, $P\{t \geq (a+s)\} = P_2$ and $P_1 + P_2$ for $m = 20$ and $o < \frac{s}{n} < 2$.

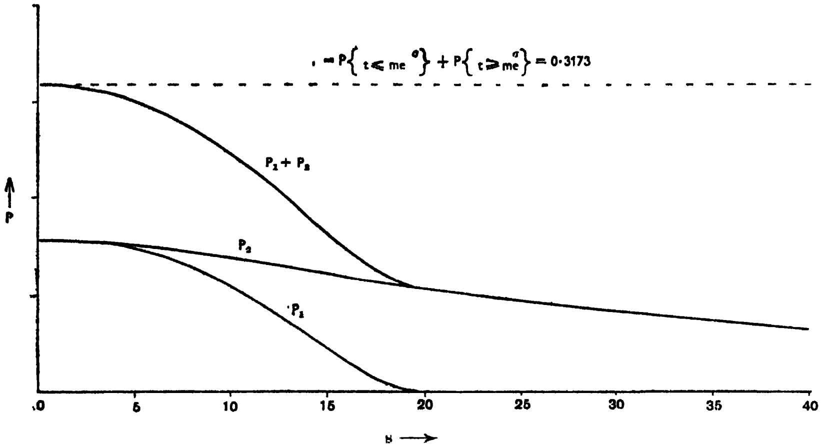


CHART C.—Giving $P\{t < (a-2s)\} = P_1$, $P\{t > (a+2s)\} = P_2$ and $P_1 + P_2$ for the values $m = 20$ and $o < \frac{s}{n} < 2$.

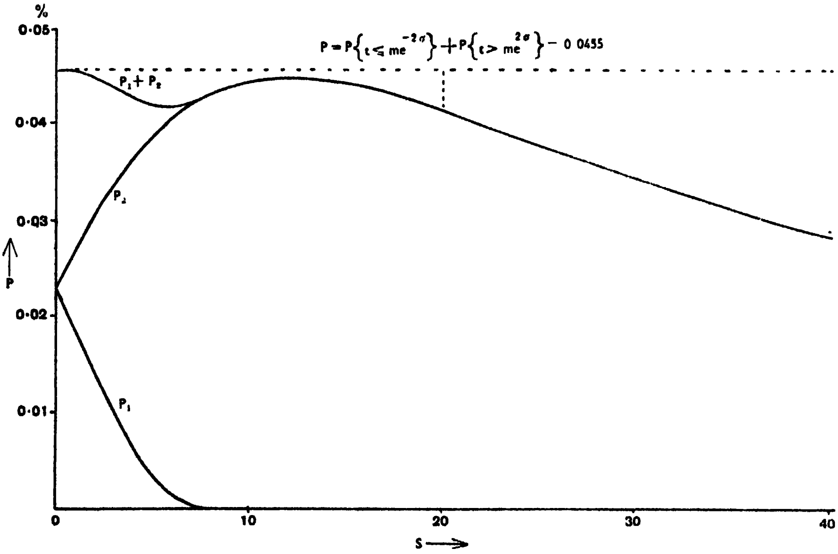
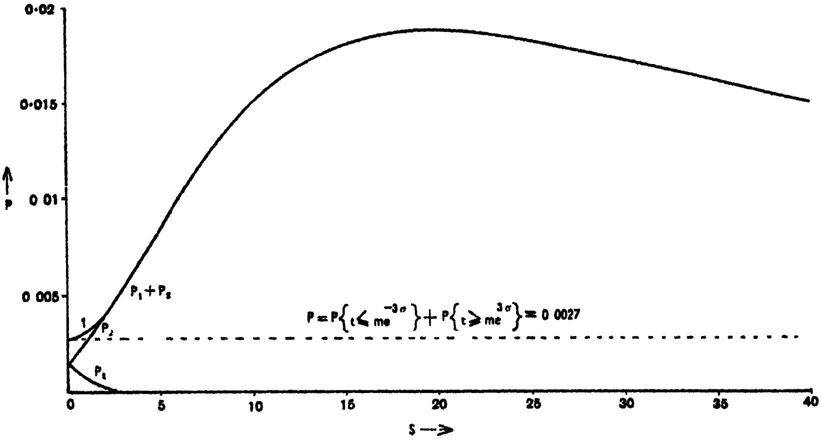


CHART D.—Giving $P\{t < (a-3s)\} = P_1$, $P\{t > (a+3s)\} = P_2$ and $P_1 + P_2$ for values of $m = 20$ and $o < \frac{s}{a} < 2$.



The two probability values become more equal as $^{s}/_{a}$ tends to *zero*. In Chart B., i.e. where $n = 1$, the normal theory value over-estimates the actual probability and this discrepancy increases with increasing values of $^{s}/_{a}$. P_1 rapidly approaches zero and the two probabilities are only approximately equal in the neighbourhood of $^{s}/_{a} = 0$, i.e. where the logarithmic distribution approaches the normal curve.

In Chart C, i.e. $n = 2$, B_1 becomes zero very rapidly but $P_1 + P_2$ remains approximately equal to P over a wide range of values of $^{s}/_{a}$. The normal theory provides a good approximation for P, when the values of the coefficient of variability ($100^{s}/_{a}$) are below *100 per cent*. This is very important from a practical point of view. The probability of getting deviations from the mean greater than twice the standard deviation is near the 5 per cent. value which forms a critical value in test criteria. Hence for $n = 2$ no serious error will follow when the normal theory is applied to a logarithmic distribution to estimate P, provided $^{s}/_{a}$ is below 1.

When $n = 3$ it follows from Chart D that P_1 becomes *zero* when $^{s}/_{a}$ is still extremely small whereas P_2 increases very rapidly with increasing values of $^{s}/_{a}$ and at reasonably small values of $^{s}/_{a}$, P_2 becomes many times greater than the normal theory value $P = 0{\cdot}0027$. The discrepancy between the normal theory value P and the probability for the logarithmic distribution is such that for reasonable values of $^{s}/_{a}$, $P = 0{\cdot}0027$ considerably underestimates the actual probability $P_1 + P_2$, as calculated from the logarithmic curve.

The skewness of the logarithmic curve is demonstrated by the P_1 and P_2 curves on the above charts. P_1 rapidly tends to zero as n is increased and becomes zero when ns is equal to or greater than a.

8. MATERIAL.

The data in the present study, which intends the application of the logarithmic function (2) to observed frequency distributions in fibre thickness measurements, were obtained by two different sampling methods. Tht first group of samples, Group A, was taken by the method of practice at the Onderstepoort Wool Laboratory. This method is described in a previous paragraph. The two samples of Group B were obtained by mounting a small sample of stretched fibres on a slide for measurement.

The samples of Group A figure in an independent investigation but were placed at the author's disposal for the purpose of this study. There were altogether six samples from each of a fine, medium and a strong wool. These wool classes were selected from a large number of fleeces and ultimately sampled.

In the second group of samples every fibre was measured once only, in order to eliminate variation "within" fibres. The thicknesses in this group were also measured on an anti-logarithmic scale of which the scale divisions are proportional to numbers with equally spaced natural logarithms (Chart E). Thus when the histogram which represents observed frequencies is drawn on a logarithmic base the group intervals are equal and the logarithmic function (2) becomes normal. An image of the magnified scale is given below. (Chart E.)

9. PRESENTATION OF DATA.

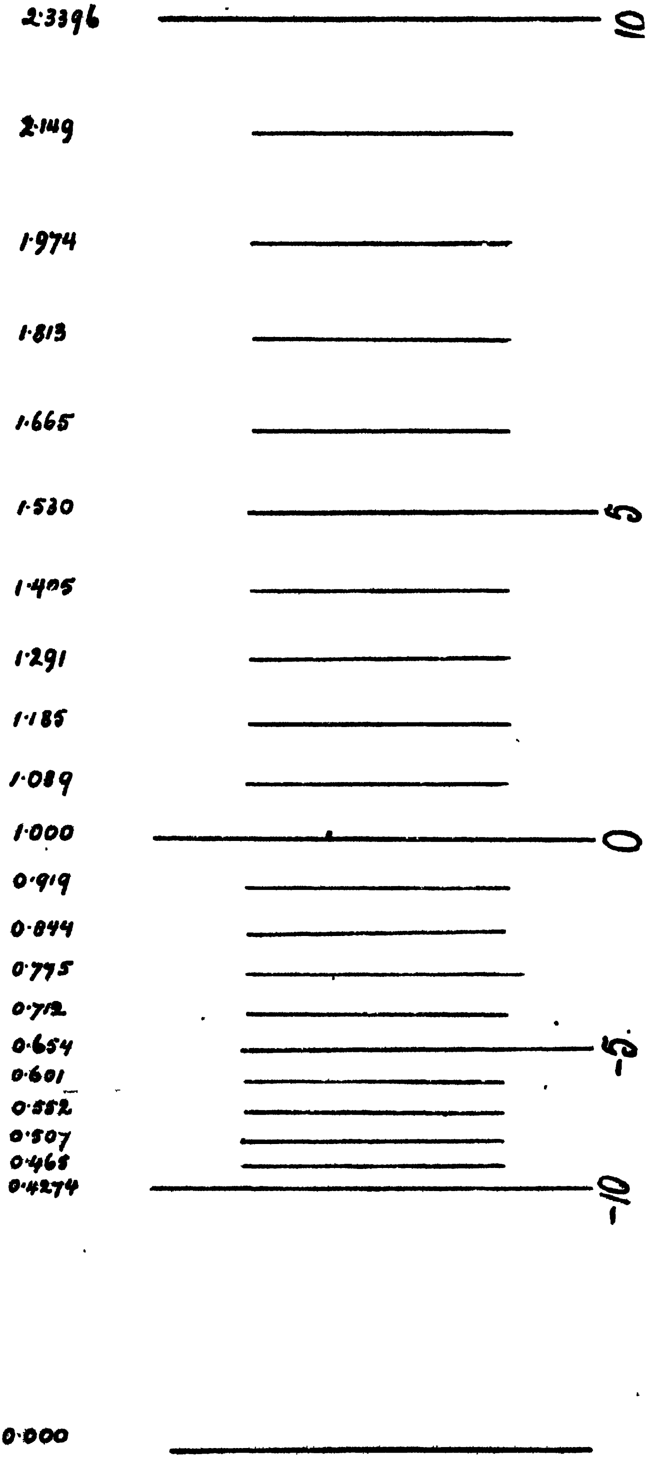
Group A.

This group of observations was taken from three different thickness classes and is presented accordingly in three separate tables. Together with each observed sample are given in adjacent columns the expected frequencies from both a normal and a logarithmic population, with the estimated mean and deviation coefficients below the respective columns. For the normal theory the standard deviations and coefficients of variability are both given but for the logarithmic distributions only the measure of percentage deviation is shown. The values of the ordinary coefficients of variability are about 20 per cent. and therefore approximately 0·2 per cent. greater than the coefficients of relative variability as shown by the tabulated values for $^s/_a$ and σ in paragraph 3, Table I.

The agreement between "observed" and "expected" frequencies is measured by χ^2. The relative merits of the two distributions, normal and logarithmic, as the parent population, may be judged from the respective probabilities. These probabilities, given in the last row of the tables, refer to the chances of obtaining the samples from the respective theoretical distributions.

The logarithmic nature of the frequency distributions of fibre thickness measurements is further illustrated by Figures 1, 2 and 3, where the frequency histograms of the data in Tables III, IV and V respectively and the best fitting normal and logarithmic curves are produced. The observed distributions in these graphs are typical of observed distributions of fibre thickness measurements.

CHART E.—The anti-logarithmic Scale.



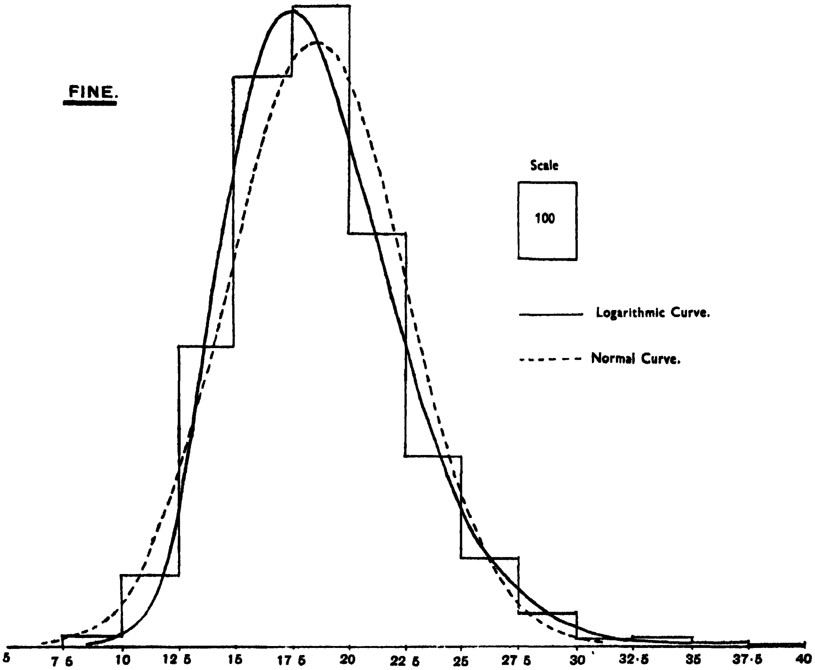


Fig. 1.—The Frequency Distribution of 3005 Fibre Thickness Measurements of a Fine Wool (μ).

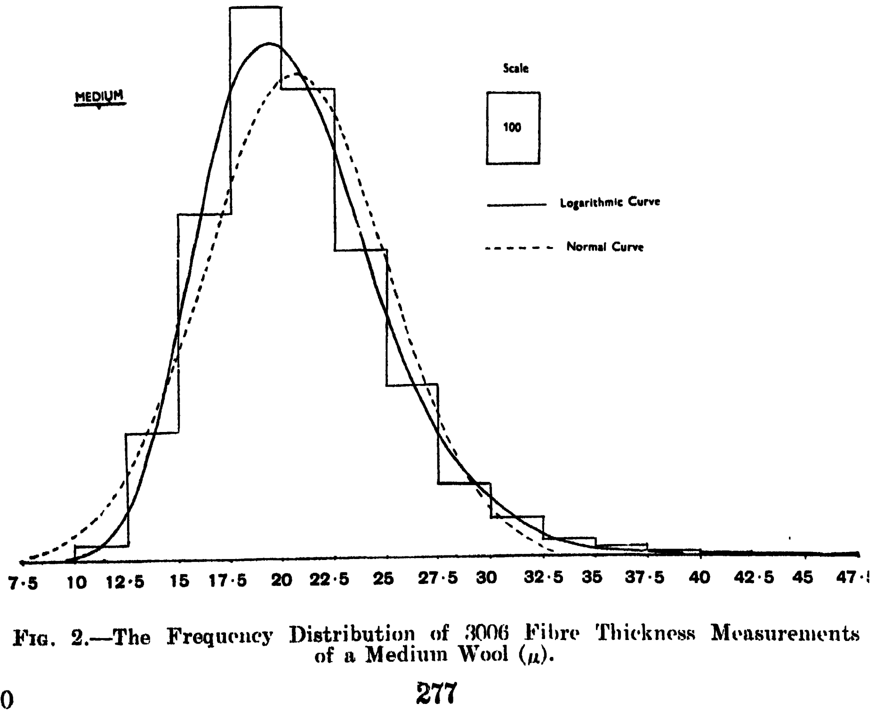


Fig. 2.—The Frequency Distribution of 3006 Fibre Thickness Measurements of a Medium Wool (μ).

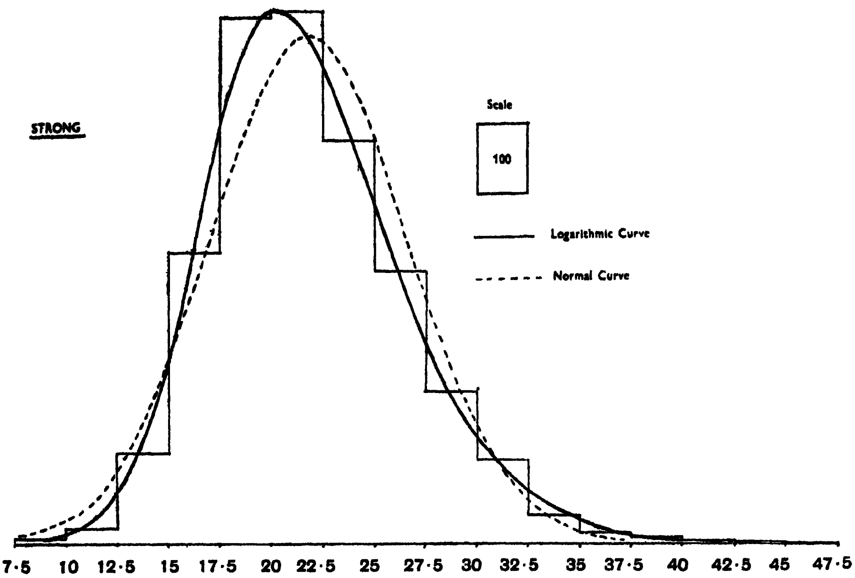


Fig. 3.—The Frequency Distribution of 3459 Fibre Thickness Measurements of a Strong Wool(μ).

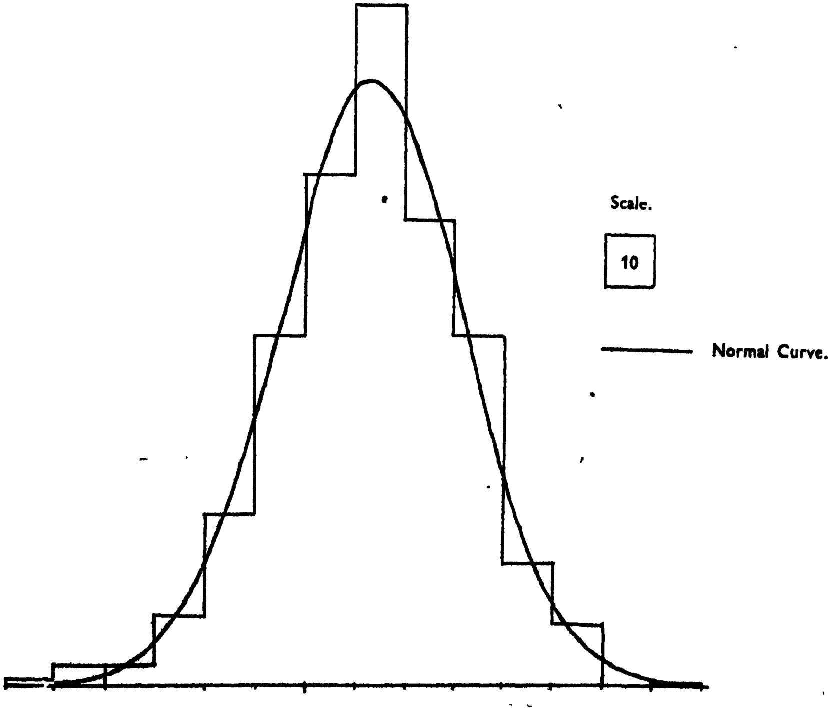


Fig. 4.—The Distribution of the Natural Logarithms of 557 Fibre Thickness Measurements.

Group B.

The two samples in this group were obtained from slides of stretched fibres, representing an ordinary shoulder sample and a sample of tops respectively. These measurements were taken by two measuring scales, the ordinary scale with a group interval of 2·5 μ [Table VI (*a*)], and an anti-logarithmic scale (Chart E) with a difference of 0·085 between the natural logarithms of consecutive divisions [Table VI (*b*)]. Unit distance on this latter scale was adjusted at approximately 20·5 μ for the shoulder sample and 21 μ for the tops. This adjustment of the magnification to obtain the precise value in μ which corresponds with unity on the anti-logarithmic scale, needs great care. Measurements by the ordinary scale in the eyepiece are not affected by alterations in magnification but for a loose scale the degree of magnification is essential for the ultimate conversion of coefficients into units of μ.

The logarithmic distribution of the ordinary measurements and the normality of the logarithmic measurements are clearly shown by Table VI. The normality is further illustrated in the cases of the shoulder sample, by Fig. 4, where the observed frequencies and best fitting normal curve for the logarithmic measurements for the shoulder sample are shown.

10. DISCUSSION.

When the normal theory is considered the apparent deficient number of relatively thin fibres and excessive number of thick fibres are obvious. This is shown by all samples and illustrated by the graphs in Figures 1, 2 and 3. The observed distributions are decidedly skew and on the whole by no means normal. The probabilities for χ^2 in the case of the normal curve are almost throughout highly significant and in the majority of cases so small, less than 0·001, that the values are not given in the tables. When the limit for significance is taken at the one per cent. probability level it is seen that there are only two samples, (3) and (31) in Table III, none in Table IV, one (22) in Table V, and one (the shoulder sample) in Table VI (*a*), which can reasonably be assumed as random samples from a normal population. When the 5 per cent. probability level is taken not a single sample can be assumed to come from a normal population.

Considering, however, the assumption that these samples are taken from logarithmic populations the position is reversed and the agreements are on the whole within the ranges of reasonable expectation. The general trend of the logarithmic curve closely follows that of the observed histogram as shown by Figures 1, 2 and 3. The probabilities for χ^2 vary between 0·91 (Table III, sample 19) and 0·011 (Table IV,, sample 4). None of the samples disagree significantly with the logarithmic theory when the one per cent. probability level is assumed. For a 5 per cent. probability level there are only three samples (4, 6 and 10) in Table IV, and none in the other tables for which the χ^2 values are significant. In this connection it is interesting to note that when modal frequency is grouped with the

TABLE VI.

Frequency Distribution in Fibre Thickness.

Group Interval (μ).	(a) Ordinary Scale.						Group Interval. (Mid. Pts.)	(b) Logarithmic Scale.			
	(1) Shoulder Sample.			(2) Tops.				(1) Shoulder Sample.		(2) Tops.	
	Observed.	Normal.	Logarithm.	Observed.	Normal.	Logarithm.		Observed.	Normal.	Observed.	Normal.
10 —12·5	—	—	—	1	—	—	—6·5	—	—	2	—
12·5—15	1	—	—	10	16·3	9·4	—5·5	—	—	—	—
15 —17·5	4	9·4	4·5	27	33·0	37·0	—4·5	1	—	9	—
17·5—20	29	29·5	30·7	86	62·3	75·0	—3·5	4	—	8	20·2
20 —22·5	91	74·1	89·6	93	85·1	89·0	—2·5	4	5·7	27	25·4
22·5—25	125	124·3	138·1	75	84·1	78·9	—1·5	14	15·1	35	42·1
25 —27·5	151	139·6	132·1	40	60·5	50·3	—0·5	34	38·6	64	57·6
27·5—30	85	105·0	88·7	36	31·0	27·0	+0·5	69·5	74·1	70	70·2
30 —32·5	47	52·6	45·3	14	11·6	12·6	1·5	101·5	107·9	55	61·0
32·5—35	16	17·7	18·7	3	3·8	8·5	2·5	135·5	119·0	49	47·0
35 —37·5	6	4·6	9·4	2	—	—	3·5	92·5	99·3	26	29·8
37·5—40	1	—	—	1	—	—	4·5	70	62·8	20	15·7
40 —42·5	—	—	—	—	—	—	5·5	25	29·9	7	10·2
42·5—45	1	—	—	—	—	—	6·5	13	14·5	1	—
	—	—	—	—	—	—	7·5	—	—	1	—
	—	—	—	—	—	—	8·5	1	—	—	—
Total	557	556·8	557·1	388	388·3	387·7	—	567	566·9	374	373·9
Mean	—	25·57	25·27	—	22·4	22·0	—	—	2·35	—	0·642
S.D.	—	3·87	—	—	4·33	—	—	—	3·504	—	5·145
Percentage deviation	—	15·2	15·0	—	19·3	19·1	—	—	—	—	—
χ^2	—	14·9	5·3	—	23·4	10·8	—	—	7·6	—	5·8
Degree of freedom	—	6	6	—	6	6	—	—	7	—	7
P(χ^2)	—	0·021	0·51	—	—	0·10	—	—	0·37	—	0·56

one following it, the significance is removed in all three cases. So for instance the probabilities for samples 4 and 10 (Table IV) become 0·42 and 0·85 respectively.

The distributions of Table VI (a) are in close agreement with those in the first three tables in that the normal theory does not seem to account for the observed distributions while the logarithmic theory supplies a good " fit " for both samples. Both methods of sampling, therefore, gave distributions which conform with the logarithmic function. This fact is further illustrated by the distributions in Table VI (*b*). The latter observations were obtained by using the anti-logarithmic scale as a measuring rule. The χ^2 values for these samples are in agreement with the assumption that they are taken from a population which becomes normally distributed when the logarithms of the values are taken.

In view of all these samples it appears reasonable to take the logarithms of wool fibre diameter measurements for purposes of statistical analysis. When this is done the arithmetical mean is replaced by the geometrical mean and since the latter is for fibre thickness measurements always slightly less than the former, it is advisable to give the quality numbers and class standards in terms of both means. The geometrical mean has various advantages for this purpose, being nearer the modal value and equal to the median. When the arithmetical mean is used to classify wool it is possible that the greater majority of fibres may actually fall in a lower class, which is obviated by the use of the geometrical mean.

11. SUMMARY.

The logarithmic nature of distributions in wool fibre thickness measurements has been suggested by the constancy of the coefficient of variability in previous work.

The distribution of a variable, the logarithm of which is normally distributed, is discussed.

The application of what is in the text called the logarithmic function to 18 different samples is given and the " fit " compared with that of the normal distribution.

Two further samples, which were also measured by an anti-logarithmic scale, are included and show that the logarithms of fibre thicknesses are normally distributed.

The logarithmic nature of the distributions of fibre thickness measurements and the normality of the logarithms of such measurements are illustrated by Figures 1, 2, 3 and 4 respectively.

It is suggested that the logarithms of fibre thickness measurements be used for statistical analysis. This would mean that the arithmetical mean is to be replaced by the geometrical mean to represent average fibre thickness.

12. ACKNOWLEDGMENTS.

The Author wishes to express his indebtedness to Dr. V. Bosman of the Wool Section for permission to use the data given in Tables III, IV and V and to Mr. C. M. van Wyk for kindly supplying the said data, which were taken in the Wool Section under his supervision, and to Mr. D. F. du Toit who assisted in the computations.

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Protozoal Diseases.

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A New Piroplasm (*Sauroplasma thomasi* n.g., n.sp.) of a Lizard (*Zonurus giganteus*, Smith).

By P. J. DU TOIT, Section of Protozoology and Virus Diseases, Onderstepoort.

UNTIL recently "piroplasms" had been observed in mammals only. Balfour, in 1907, had seen bodies resembling piroplasms in the blood of fowls suffering from spirochaetosis in the Sudan, but he failed to recognize their true nature. It was only in 1928 that Carpano first described a piroplasm of domestic fowls under the name *Aegyptianella pullorum*. The finding of what appeared to be a near relative of the mammalian piroplasms in birds showed that these unpigmented, endoglobular blood parasites had a wider distribution than was at first supposed.

In 1935 Brumpt and Lavier described a piroplasm (*Tunetella emydis*) from the blood of a tortoise (*Emys leprosa*), thus extending the known range of distribution of these parasites from the warm-blooded mammals and birds to the cold-blooded reptiles.

In the present article a parasite, which also seems to be a true piroplasm, will be described from the giant girdle-tailed lizard, *Zonurus giganteus*. It would seem, therefore, that the occurrence of piroplasms in reptiles is more general than was supposed at first.

Actually there is nothing very surprising in this finding of piroplasms in reptiles seeing that ticks, the sole transmitters of piroplasmosis, attack all classes of vertebrates living on dry land.

Before proceeding to the descrption of the parasite it may be useful to make a few remarks about the parasites of the red blood corpuscles of reptiles in general.

REVIEW OF LITERATURE.

For the purpose of this article it is unnecessary to review the literature in any detail. Suffice it to refer to the excellent summaries in some of the more recent text books on protozoology, such as that of Wenyon (1926) and Doflein-Reichenow (1929), and to discuss only the few articles which are of special interest to our subject.

All endoglobular blood parasites of reptiles (and other vertebrates) belong to the Sporozoa.

This large class is again sub-divided into many orders and families, but there is considerable difference of opinion between protozoologists about the details of this classification. These differences need not concern us here. For our purpose we may assume the validity of the families Haemoproteidae, Plasmodidae, Haemogregarinidae, and Piroplasmidae (or Babesidae), to which most of the parasites in which we are now interested, belong.

The members of the two first-named families are characterized by the production of pigment in the host cells (the red blood corpuscles). The parasites belonging to this group can therefore be recognized fairly easily and cannot readily be confused with the piroplasms which are unpigmented. A large number of species of *Haemoproteus* and *Plasmodium* has been described from reptiles.

The haemogregarines are a somewhat ill-defined group containing, according to some authors, the single genus *Haemogregarina*, according to others, a considerable number of genera and even families belonging to the sporozoan order Adeleidea. Again this difference of opinion need not detain us. It is of interest though to note that a very large number of haemogregarines occurs in reptiles and particularly in lizards. The life-histories of many of these species are well known and their blood forms can easily be distinguished from piroplasms.

Of the piroplasmidae only one member has thus far been described in reptiles. Brumpt and Lavier (1935, b) observed in the blood of a tortoise (*Emys leprosa*) from Tunis an endoglobular parasite which they called *Tunetella emydis*. This parasite occurred in three forms: (1) large forms, 2-5μ in diameter, round, oval or lobulated in shape, with one to three chromatin granules; (2) small forms, less than 1μ in diameter, single or in groups; and (3) small inclusions, arranged in rosettes or in rows, appearing as basophilic spots, but without any chromatin granules.

The authors discuss the relationship between these different forms and come to the conclusion that they probably represent developmental stages of the same parasite. An attempt to transmit the parasites from the infected tortoise to fourteen other tortoises of the same species failed; the authors suspect that these specimens were immune.

Brumpt and Lavier then discuss the relationship between this parasite and other known parasites, particularly *Aegyptianella pullorum*, and they conclude that their parasite does not belong to this genus but should be placed in a separate genus.

One further parasite of reptiles, thus far recorded in the literature, can be said to have a resemblance to the piroplasms. I am referring to a blood parasite of a gecko, *Tarentola mauritanica*, described by Chatton and Blanc, (1914, 1916) under the name *Pirhemocyton tarentolae*. Three out of about forty geckos caught in the neighbourhood of Matinata and Metlaoui in Tunis were found to be infected.

When the fresh blood was examined the authors noticed, not the parasites themselves, but globular, albuminous, refractive bodies in the red blood corpuscles. One of these bodies seemed to be present whenever there was a parasite in the cell. There was also a direct relation between the size of the parasite and that of the globular body but otherwise the two objects seemed to be in no way related.

The parasites themselves varied from small anaplasmoid bodies of about 1μ in diameter, to larger round, oval or pear-shaped bodies 3 to 4μ in diameter. The authors distinguish anaplasmoid forms, some of which have a clear zone around them; spherical forms with a chromatin dot in the centre of the clear cytoplasm; other spherical forms with the chromatin more diffuse; and a variety of other forms. Some of these parasites are closely connected with the nuclei of the host cells; in fact in some cases a chromatin thread connects the nucleus of the parasite with the nucleus of the red blood corpuscle; and in others the parasite seems actually to be extruded from the cell nucleus.

The authors did not observe any multiplication forms of the parasites, and suggest that, as in *Theileria parva*, multiplication does not take place in the peripheral blood. (Spleen and marrow were not examined.) The relationship of these parasites to other known groups is discussed, and the authors come to the conclusion that there is no close connexion with the piroplasms.

The same conclusion is reached by Brumpt and Lavier (1935, a) in regard to a very similar parasite, *Pirhemocyton lacertae*, which they found in a green lizard. *Lacerta viridis*, obtained from Italy. They found the same forms of the parasite but did not observe the globular bodies which were so prominent in Chatton's and Blanc's cases. Brumpt and Lavier succeeded in transmitting the infection to another lizard of the same species: the incubation period was six days.*

PERSONAL OBSERVATIONS.

The parasites about to be described, were found in the red blood corpuscles of a lizard *Zonurus giganteus*, Smith. The first infected specimen was caught by Dr. A. D. Thomas of the Onderstepoort Veterinary Research Institute during a tour in connexion with the Zoological Survey of South Africa, on the farm Jacobsdal near the village of Wesselsbron, in the Orange Free State, in January, 1936. During a subsequent tour in November, 1936, Thomas found another infected specimen on the town lands of Odendaalsrust in the Orange Free State. At the time only blood smears were collected, so that it was impossible later on to examine either the fresh blood or other organs for possible developmental stages of the parasites.

---

* Mention should also be made of a parasite of the blood of fish (*Cottus bubalis* and *Cottus scorpius*) described by Henry (1910 and 1913) under the name *Haemohormidium cotti*. The parasites have a resemblance to piroplasms although Henry himself does not include them in this group but suggests some relationship to the haemogregarines.

The degree of infection of the blood of the first specimen was somewhat heavier than that of the second, and the first contained more divisional forms of the parasite than the second, but otherwise the two may be considered as identical.

The blood of both specimens showed a large number of infected blood cells. In the first specimen a count of 500 cells revealed a percentage infection of 56. In the second specimen the corresponding figure was 43 per cent. In the great majority of cases an infected erythrocyte contains a single parasite. Two parasites in a blood cell are very rare, and more than two were never seen.

The parasites are small in comparison with their host cells. Accurate measurements revealed a variation between 0·6μ and 4 5μ in diameter or length of the parasite. The majority are between 1μ and 2·5μ in diameter. (The red blood corpuscles of the host measure on an average about 20 x 10μ.)

The micro-photographs and drawings accompanying this article will give an idea of the variation in size and shape of the parasites.

The smallest forms are either anaplasmoid bodies consisting of a chromatin granule or small ring-shaped bodies. It would almost seem as if the ring-shaped bodies, in some cases at least, arise out of the anaplasmoid bodies by the appearance and gradual enlargement of a "vacuole" or "lumen" in the centre of the chromatin granule. However, in other cases the ring-shaped bodies are formed as such, as we shall see below.

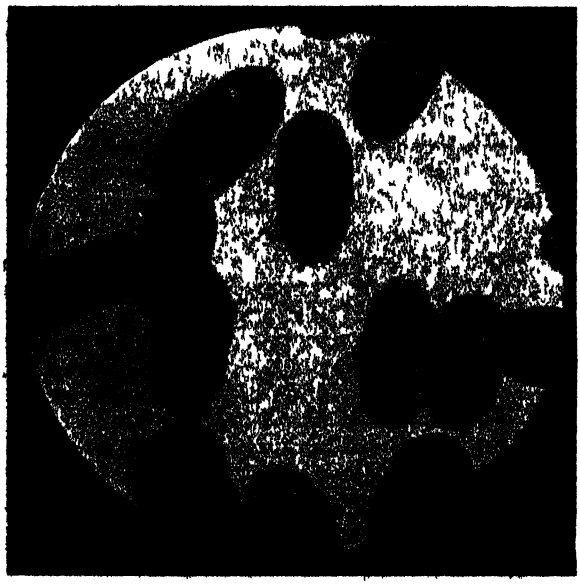


Fig. 1 (a).

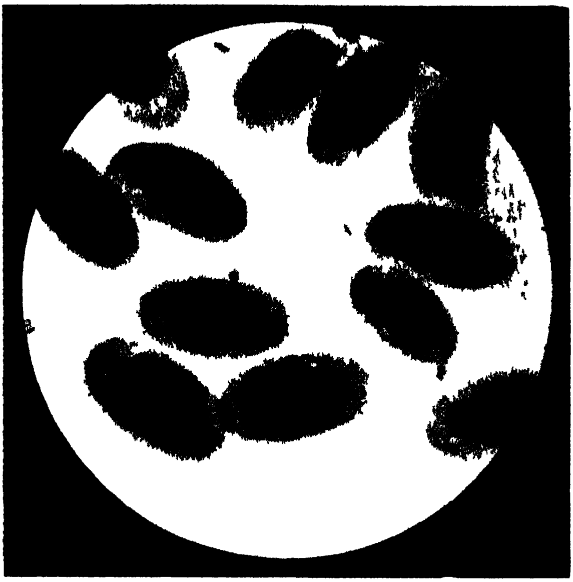


Fig. 1 (b).

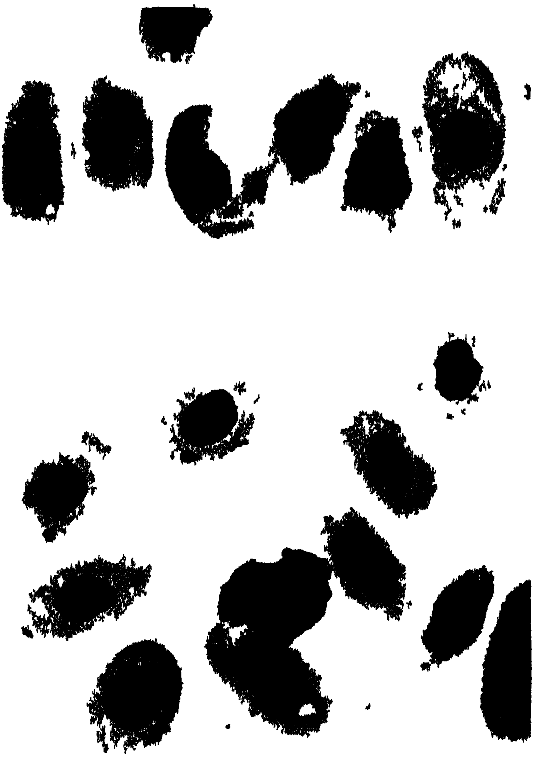


Fig. 1 (c).

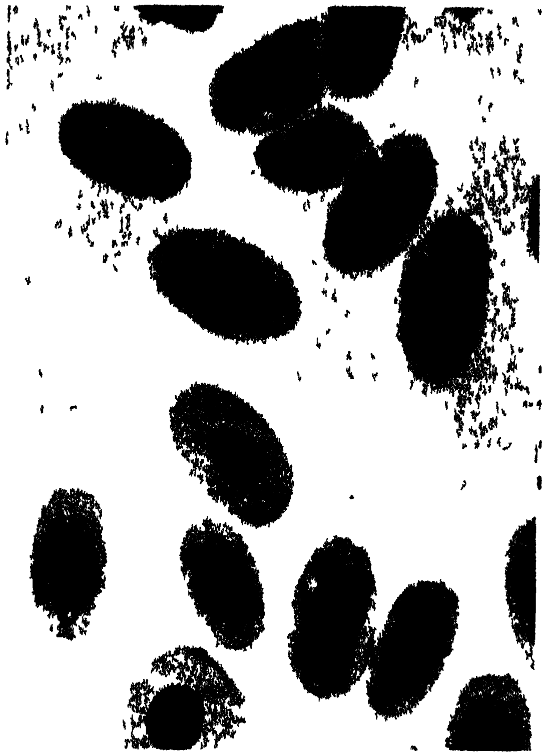


Fig. 1 (*d*).

Fig. 1 [(*a*), (*b*), (*c*) and (*d*)].—*Squroplasma thomasi* in the blood of *Zonurus giganteus*. Note the large, clear "vacuoles" in some of the parasites. Magnification 1000×.

The great majority of parasites are more or less ring-shaped. Some are almost perfectly spherical, whereas others are irregular in outline. In most of the parasites the periphery of the ring is clearly marked, partly by well-stained cytoplasm, partly by nuclear material; however, in some the chromatin is only faintly visible. Frequently the nucleus sits like a cap on one side of the spherical parasite, giving it the appearance of a signet ring. In other instances the nuclear material almost or completely surrounds the cytoplasmic body.

The central portion of these parasites is almost invariably clear. Whether an actual vacuole is present cannot be stated with certainty, but this is the impression which is conveyed. Because of their clear centre, which lets the light through, these parasites are very easily recognized in stained blood smears.

In some parasites the degree of staining seems to be more intense, probably because of a relatively larger amount of nuclear matter. Such parasites appear as dark masses of round or irregular shape. Sometimes a small area of clear cytoplasm is visible.

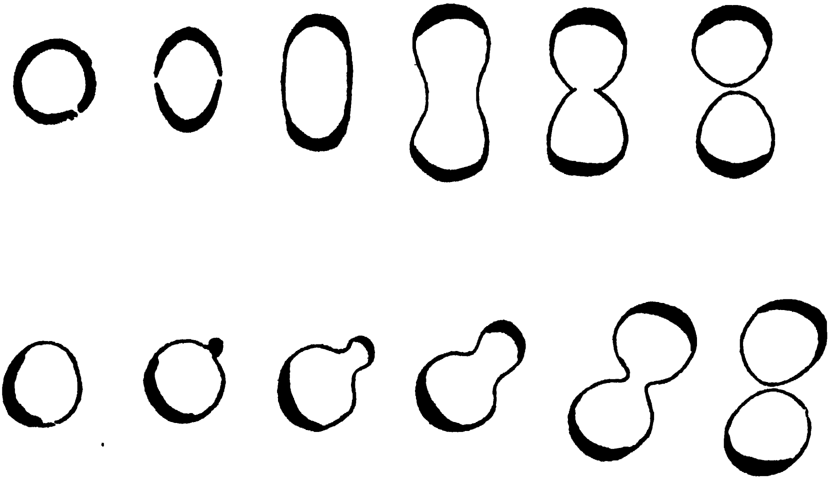


Fig. 2.—Diagrammatic illustration of the process of multiplication in *Sauroplasma thomasi.* In the upper row division takes place by binary fission; in the lower row by a process of budding.

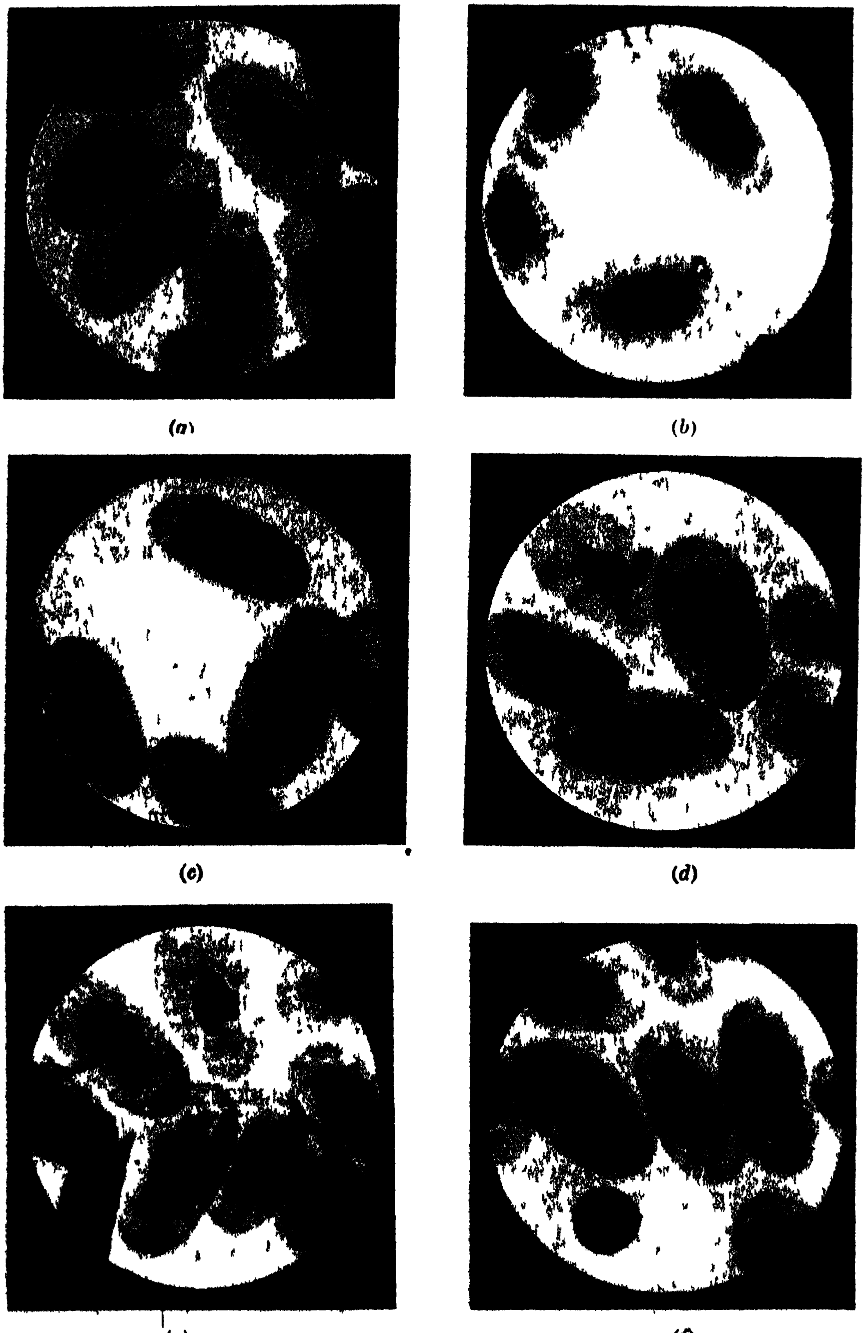
The process of multiplication in these parasites is interesting. Apparently multiplication invariably takes place by a process of division into two. In the accompanying microphotographs and drawings the process is clearly illustrated. It would seem that there are two methods by which this division into two is achieved. In the first (see Fig. 2) the spherical parasite elongates; the nuclear material concentrates at the two opposite ends. Then a constriction appears in the middle of the elongated parasite. This constriction continues until two separate and approximately equal daughter cells are formed:

The same result is achieved by the second method which is a process of budding. On the outer surface of the spherical parasite a bud appears, which at first seems to consist of solid chromatin. Soon more material is extruded at this point so that the bud acquires a lumen. The bud continues to grow until it has approximately the same size as the mother cell when, by a process of constriction, the two cells become separated. It should be noted that this process is very similar to that by which multiplication takes place in many species of piroplasms.

These two processes may be referred to as " binary fission " and " budding " respectively (see Figs. 3 and 4).

DISCUSSION.

The parasites here described seem definitely to belong to the family Piroplasmidae (or Babesidae). Their intracorpuscular situation, the absence of pigment, their mode of multiplication, their size and shape are all features which characterize the piroplasms.



(*a*) (*b*)

(*c*) (*d*)

(*e*) (*f*)

Fig. 3.—A series of microphotographs of *Sauroplasma thomasi* illustrating the process of “budding”. (*a*) Appearance of small bud consisting of solid chromatin. (*b*) Enlargement of bud. (*c*) Appearance of lumen in bud (*d*) Growth of daughter cell. (*e*) Two cells practically the same size. (*f*) Two daughter cells side by side. Magnification 1,250 ×.

When these parasites were first observed in the blood of the lizard it was thought that they might belong to the genus *Aegyptianella* of birds However a closer study immediately revealed many points of difference The most important of these is the absence of any process suggesting schizogony in the lizard parasite

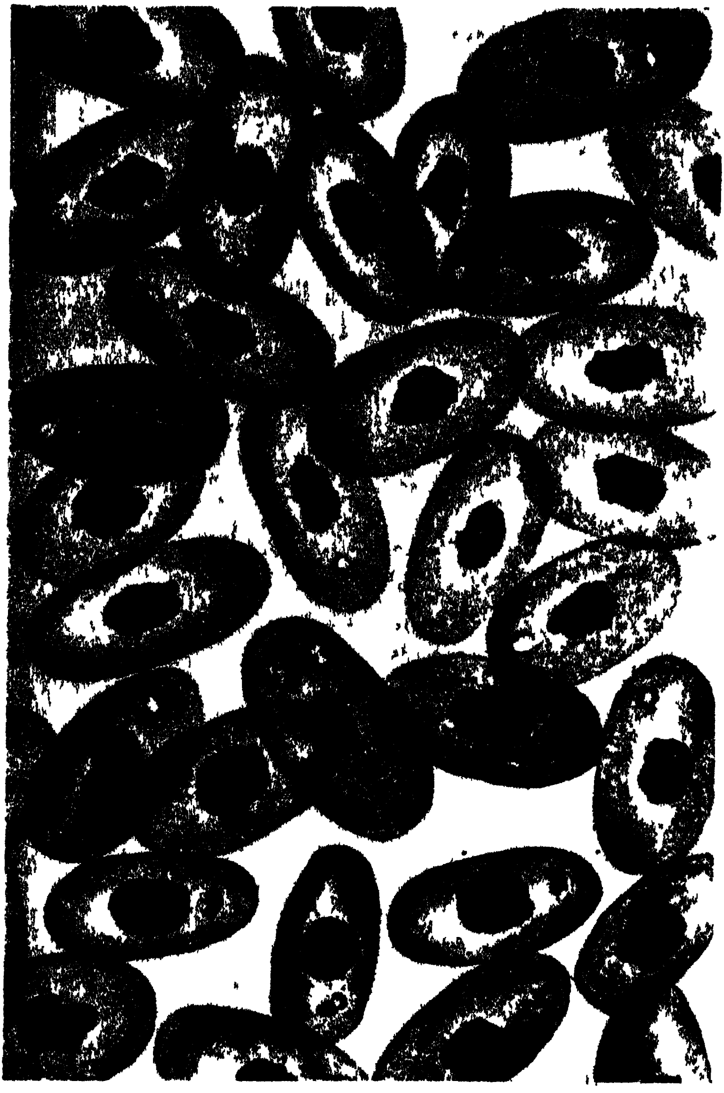


Fig 4 —Composite drawing of red blood corpuscles of *Zonurus giganteus* infected with *Sauroplasma thomasi* Note the divisional forms, both the process of "budding" and that of "binary fission are illustrated

A comparison with *Tunetella emydis* of the tortoise also shows that the two parasites differ from each other in many respects. The large forms of *Tunetella* are much larger than the lizard parasites; and the small basophilic spots which form such an important feature of the infection in the tortoise have no counterpart in the lizard. Nothing resembling the "pseudo-schizogony" described by Brumpt and Lavier has been seen in the lizard.

Attention should here be directed to an article by Coles appearing in this Journal, in which a parasite of fowls is described that certainly shows a close resemblance to our parasite of the lizard. A comparison of the microphotographs of Coles' parasite with those appearing in this article will reveal the similarity.

The question whether these piroplasms of the lizard can be placed in any existing genus of the family Piroplasmidae (Babesidae) is difficult to answer in the present state of our knowledge. Many features seem to separate this species from the known piroplasms of mammals, and the differences which separate it from the genus *Tunetella* have already been referred to. It is therefore proposed to create a new genus for this parasite and to call it, in honour of Dr. A. D. Thomas who discovered the infection in the lizard, *Sauroplasma thomasi*.

The genus *Sauroplasma* could be defined as follows: Small, round or irregularly shaped unpigmented parasites of the red blood corpuscles of lizards. The typical form is that of a ring or signet ring with a large vacuole. Multiplication takes place by binary fission or by a process of budding.

It may be added that the two lizards in which these parasites were found appeared to be completely healthy. The infected blood showed no signs of anaemia. It would thus appear that in this case, as in so many other parasitic infections of wild animals, the parasites have no pathological effect on their host, but live in perfect harmony with the host.

A few further specimens of *Zonurus giganteus* which were examined showed no infection of the blood.

In regard to the mode of transmission of these parasites nothing is known at present. No ticks were seen on the specimens that were caught. Nevertheless it is regarded as most likely that *Sauroplasma*, like all other piroplasms, is transmitted by ticks.

SUMMARY.

A parasite is described which infects red blood corpuscles of the giant girdle-tailed lizard *Zonurus giganteus*, Smith. The relationship of this parasite to other organisms is discussed and the conclusion is reached that it represents a new genus of the sporozoan family Piroplasmidae (Babesidae). The name *Sauroplasma thomasi* is proposed for it.

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A New Blood Parasite of the Fowl.*

By J. D. W. A. COLES, Section of Poultry, Onderstepoort.

WHILE in the United States of America in 1935, the author examined several blood smears of fowls showing anaemia due to different causes. In smears of two fowls taken in New York City the parasites were found. As far as was known the fowls were raised not far from the city. Later similar bodies were encountered in a fowl blood smear received from Philadelphia. During the year following his return to South Africa the writer found on three occasions in fowl blood smears an organism morphologically indistinguishable from that seen in America.

DESCRIPTION OF THE PARASITE.

It is an intraerythrocytic organism seen so far only in the blood of the fowl. Usually almost round, it varies in diameter from $0 \cdot 5\mu$ to $1 \cdot 5\mu$, the average diameter being $1 \cdot 0\mu$. Those that are oval may be $1 \cdot 5\mu \times 0 \cdot 7\mu$.

The centre of the parasite is usually clear. The chromatin may extend right round the periphery; often, however, it is most prominent round only one half of the circumference. The chromatin is sometimes seen lumped at two or three points on the periphery. When the organism is distinctly oval, the chromatin is often aggregated at the extremities to give a bipolar appearance. Pairs of parasites are not rarely found, suggesting that division is by binary fission. Figures 8, 9 and 10 illustrate this point. Our knowledge of the mode of multiplication is still most fragmentary. No pigment has been seen.

It is very unusual to find two well separated parasites in one cell and, so far, three or more have not been observed in a single cell. In nearly all cases only one parasite occupies a cell, and it is usually situated midway between the nucleus and the edge of the cell

Parasites have rarely been seen in normoblasts. Giemsa's stain has been the only one so far employed.

---

* The organism was discovered while the author was studying as a Commonwealth Fund Service Fellow in Dr. E. V. Cowdry's laboratory in Washington University, St. Louis, Mo.

Thanks are due to Dr. Cowdry for his unfailing interest and kindness.

The heaviest infection revealed only about 7 per cent. of the red cells containing parasites. Lung smears, and sometimes heart blood smears, show more organisms than smears of the peripheral blood.

Though apparently associated with some degree of anaemia the effect of the parasite on the host is uncertain. But anyone conversant with the ravages of Aegyptianellosis in nature, and its remarkable harmlessness in the laboratory, will hesitate to declare any new parasite pathogenic or otherwise, without first amassing considerable experience of its potentialities.

THE OCCURRENCE OF A SIMILAR PARASITE IN SOUTH AFRICA.

1. In February, 1936, two dead and two live White Leghorn pullets, four months old, were received from the Johannesburg district of the Transvaal. The owner stated he had lost many fowls with similar symptoms.

Clinical Examination.—The birds were mopy and stood about, occasionally drinking water. They were pale and very emaciated and had no appetite.

Post-mortem Examination.—All were markedly emaciated. The crops contained a little slime. In one case a few oocysts of Eimeria (species not determined) were detected, but there were no lesions to warrant a diagnosis of coccidiosis. Heart blood cultures on agar and in bouillon were negative. In all cases the gall-bladder was distended to a most unusual degree with dark green viscid bile.

Only in one case did a blood smear show anaemic changes and they were very slight; this heart blood smear, however, revealed a few parasites scattered more or less in groups. The peripheral blood of the same fowl appeared to be free of parasites.

Eight days later six more sick pullets were received from the same farm. All were thin and mopy and were killed for examination. In all cases round worms and tapeworms were found to be frequent. There was, as before, no tumor splenis. All the gall-bladders were very distended with dark green viscid bile. Blood smears of all pullets showed slight anaemic changes, and three birds had parasites, especially in lung smears, but the parasites were rare.

The cause of the mortality could not be established with certainty. One naturally was inclined to attribute the deaths to helminthiasis, but the condition of the gall-bladders was remarkable, and this fact alone suggested that the parasites may have had something to do with the losses. Another disease characterised by extreme emaciation and dejection, and associated with the presence usually of very few parasites, is leucocytozoosis of the fowl. This fact should make one chary about overlooking the harm that the new parasite may be capable of producing.

2. In September, 1936, three South African Australorp chickens, two weeks old, were sent for examination from the Standerton district of the Transvaal. They were dead and partially decomposed.

Death was due to *Salmonella pullorum*. In one case a lung smear showed slight anaemia and a 5 per cent infection of the red cells with the parasite.

This is of interest, indicating the incubation period may be less than 14 days.

3. In September, 1936, fifteen dead White Leghorn chicks, one month old, were received from the Leslie district of the Transvaal. In each case the liver was enlarged and yellowish, the spleen was enlarged and dark red, the kidneys were slightly enlarged and of an ochre colour, and there was intestinal catarrh. The lesions were thus typical of fowl typhoid in the chick, and the diagnosis was confirmed in each case by the isolation of *Salmonella gallinarum* from the heart blood. Two heart blood smears showed fairly marked anaemia and the intracorpuscular parasites.

Obviously the deaths were due primarily, if not entirely, to fowl typhoid. The rôle of the protozoon could not be assessed.

DISCUSSION.

Are there sufficient grounds for considering this a new parasite? It is scarcely necessary to prove it is not *Plasmodium gallinaceum* or a Leucocytozoon. Some may suggest it is part of the life cycle of a spirochaete, but now that it is clear that the intracorpuscular bodies, described by Balfour and others in association with avian spirochaetes, are *Aegyptianella pullorum*, the suggestion loses its attraction. There is no satisfactory evidence that an avian spirochaete ever enters an erythrocyte or multiplies other than by transverse binary fission. Then again there is not likely to be an Argas infestation in New York State, and the spirochaete is transmitted by species of this genus. (An unconfirmed report of Zuelzer states that mosquitoes may transmit spirochaetes to fowls, but the epizootological evidence in most countries, including South Africa, is against this being of any importance.)

Carpano has described a Grahamella infection of the fowl, but this claim also lacks confirmation and, in any case, his description of the organism indicates clearly it is nothing like the parasite under discussion.

In the differential diagnosis the parasite of outstanding importance is *Aegyptianella pullorum*, with which we are fortunately well acquainted. Aegyptianella is generally two to four times the size of the newly found organism, and two or more are commonly found in a single erythrocyte. In fowl blood, schizonts are seen fairly frequently and the merozoites can be distinguished easily. Though a merozoite is often the same size as the new organism, it tends to be elongate, and to occur in numbers of up to ten to twenty in the red cells. Aegyptianella has been proved to be carried by *Argas persicus* and, as already mentioned, the genus Argas is of no consequence in the State of New York.

All facts considered, it seems highly probable that the parasite described here should be regarded as a new fowl haematozoon, of possibly low virulence. No name is suggested as only meagre information is available regarding multiplication, mode of transmission, pathogenicity, etc.

Though smaller, the organism bears some resemblence to the parasite *Sauroplasma thomasi* n.gen.n.sp. described by du Toit (1937) in the erythrocytes of the lizard, *Zonurus giganteus*, Smith.

SUMMARY.

An apparently new blood parasite of the fowl, found in New York and Philadelphia, has been described. A very similar, if not identical, organism occurs also in South Africa. The indications are that the parasite is not highly pathogenic.

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DESCRIPTION OF PHOTOMICROGRAPHS.

These were prepared by Mr T. Meyer in his usual careful manner.

All photographs are of fowl blood stained with Giemsa

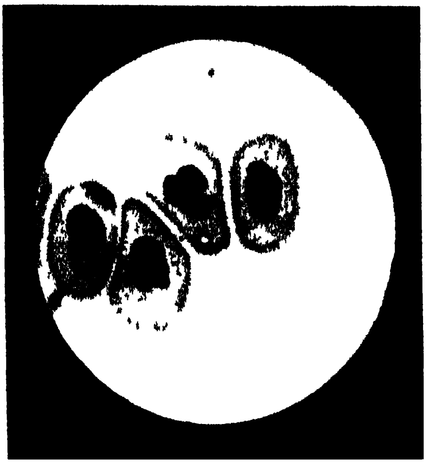

Fig 1

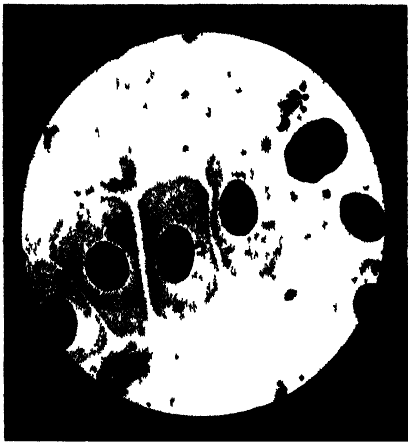

Fig. 2.

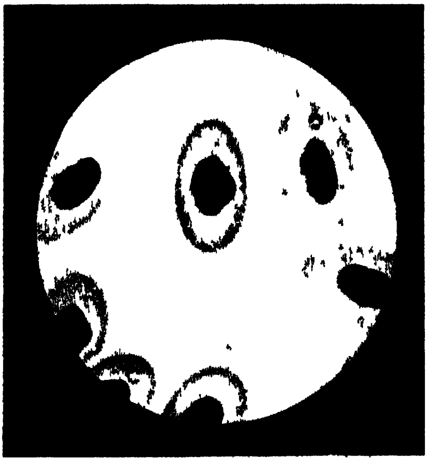

Fig 3

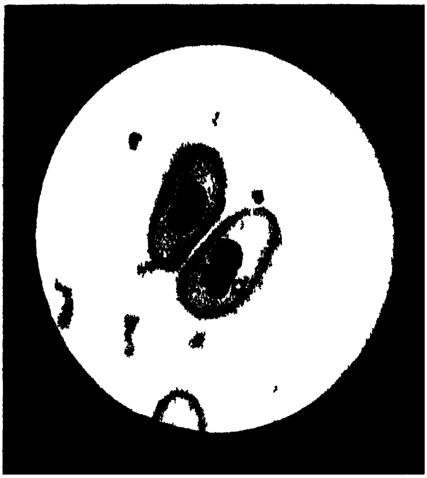

Fig 4

Fig 1.—1,400 × The largest parasite so far seen
Fig 2—1,400 × A parasite of average size
Fig. 3—1,400 × Two parasites
Fig 4—1,250 × A parasite of average size

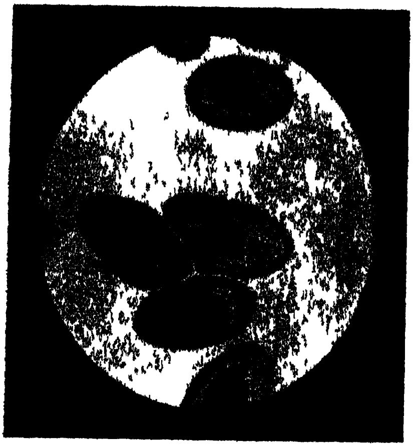

Fig. 5.

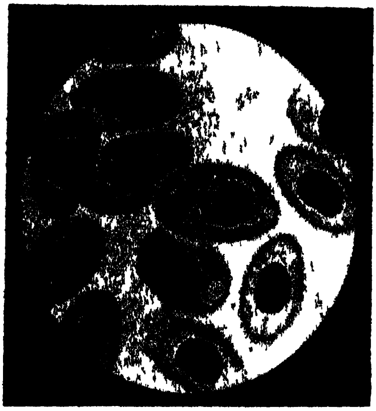

Fig. 6.

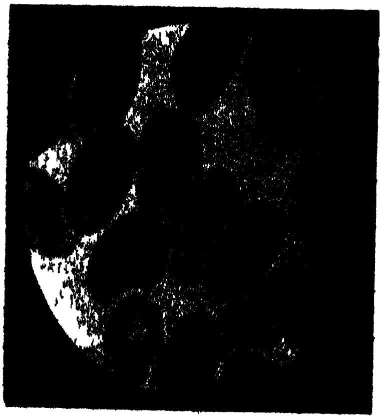

Fig. 7

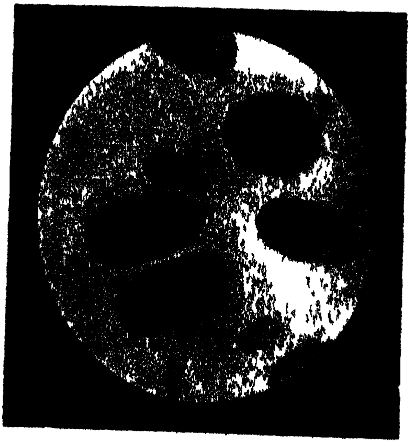

Fig. 8.

Fig. 5.—1,400 ×. A parasite of average size.
Fig. 6.—1,250 ×. A bipolar form.
Fig. 7.—1,250 ×. A smaller form and a larger form (out of focus).
Fig. 8.—1,400 ×. A parasite apparently dividing.

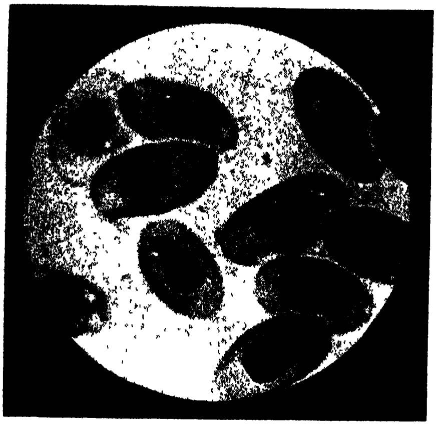

Fig. 9.—1,250 ×. Two adjacent organisms, possibly the result of binary fission.

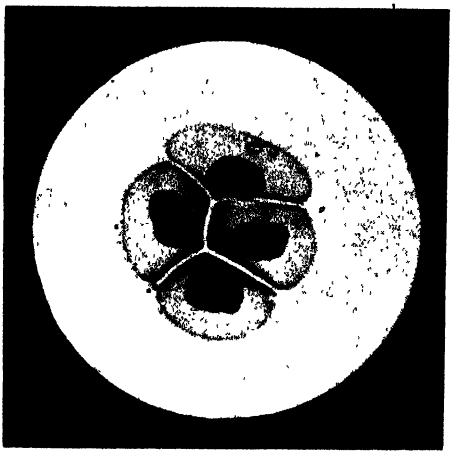

Fig. 10.—1,400 ×. Two adjacent organisms, possibly the result of binary fission.

Section II.

Parasitology.

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South African Helminths.—Part I.

By R. J. ORTLEPP, Section of Parasitology, Onderstepoort.

THE present paper is intended to be the first of a series in which the helminths, collected in connection with the zoological survey of South Africa, are described. In addition helminths which are sent in from other sources may also be included. In this communication all the helminths except one, namely *Hyracofilaria hyracis* gen. and sp. nov. were collected in connection with the survey.

CESTODA.

Fam. TAENIIDAE Ludwig, 1886.

Echinococcus felidis sp. nov.

Numerous examples of this parasite were collected from a lion, whose intestine was literally felted over by the parasites. Macroscopically no lesions were discernible.

The largest specimens, all of which were fixed in alcohol, were 5·5 mm. long and consisted of the most part of four segments plus a head and neck. Some specimens, however, only had three segments, but in these the last segment was not gravid, and in some specimens the beginnings of a fifth segment was just discernible at the hind end of the neck where the developing genitalia showed up as an opaque central patch. In specimens with four segments the last segment was always gravid and the third from last mature; the segment between these had lost practically all its genitalia and the uterus was clearly defined and contained immature eggs.

The head has a somewhat square outline, the suckers being prominent and occupying the corners; it is from 0·365 to 0·406 mm. broad. The suckers are somewhat circular, 0·133 to 0·157 mm. in diameter and have a depth of 0·07 to 0·075 mm. In extended specimens the rostellum is from 0·11 to 0·16 mm. high with a breadth of 0·139 to 0·174 mm. It carries a double row of hooks, numbering 32 to 46. The anterior larger hooks (Fig. 1) are from 0·037 to 0·042 mm. long measured in a straight line from the base of the handle to the tip of the blade; their most characteristic feature is the general ruggedness of the handle and guard, both showing a markedly gnarled appearance. The central axis of the handle forms practically a straight line with that of the posterior half of the blade, and the blade in most cases is not strongly arched. In the smaller hooks the handle and guard show at most only a wavy outline, and the guard is generally heart-shaped in lateral view; the axis of the posterior half of the blade is also more or less in a straight line with that of the handle. From the tip of the blade to the end of the handle the length is 0·028 to 0·035 mm.

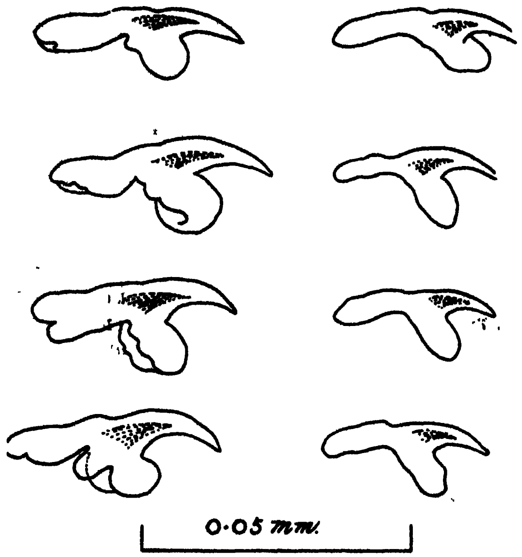


Fig. 1.—*Echinococcus felidis* sp. n. Large and small rostellar hooks.

The neck in extended specimens is generally slightly longer than broad, being from 0·26 to 0·69 mm. long, including the developing first segment which has not yet been segmented off, and from 0·29 to 0·35 mm. broad.

Mature segments are from 0·6 to 0·64 mm. long by 0·27 to 0·29 mm. broad across the level of the genital port; its anterior breadth is only about half of its posterior and about ⅔ of the maximum breadth of the segment. The genital pore, which alternates irregularly, is lodged at or just posterior of its middle. The genitalia are somewhat similar to those of the genotype, except that the anterior testes attain maturity later than the posterior one and are consequently smaller; there are from 28 to 46 testes to each mature segment, the anterior about 0·015 mm. in diameter and the posterior about 0·04 mm.; they are arranged in a single sheet to form a horse-shoe round the female genitalia, and then pass forwards on the aporal side to form a band of 3 to 4 testes broad in the central area of the segment in front of the cirrus pouch. The cirrus pouch is club-shaped and passes obliquely inwards and forwards to about the middle of the segment; it is about 0·145 mm. long by 0·05 mm. broad across the club. In gravid segments it is longer and may attain a length of 0·2 mm. with a maximum thickness of 0·1 mm.

Gravid segments are larger and may attain a length of 2·4 mm. or nearly half the length of the entire parasite, by 0·5 mm. broad; the genital aperture is found in its posterior half at about the junction of its 3rd and 4th fifths; the uterus extends throughout its whole length and shows no definite branches, instead from 15 to 20 shallow sacculations are present on either side. The onchospheres have a finely pitted shell, 0·005 mm. thick and itself is slightly oval, measuring from 0·038 to 0·039 mm. long by 0·031 to 0·034 mm. broad; the embryophore is rounded with a diameter of about 0·22 mm.

Discussion.—The parasite described above is the first record of an Echinococcus occurring in a member of the Aeluroidea in South Africa; only one other species of Echinococcus has been described from the cat tribe, namely *E. oligarthrus* (Diesing, 1863) from *Felis concolor* and *Felis yaguarundi*, S. America. It is true that *E. granulosus* has been recorded several times from domestic cats, but from the work of Lorencz (1933) it is doubtful whether this parasite is normal for this host and whether it ever attained maturity; Lorencz artificially infected 51 cats and found that this parasite developed only very slowly in this host and never attained maturity, whereas artificial infection of dogs with the same material produced egg-laying adults in 36 days.

In that the mature segment is always the third from the last, this species appears to be more closely allied to the species *E. cameroni* Ortlepp, 1934, and *E. lycaontis* Ortlepp, 1934. It may, however, be distinguished from the latter species by the rugose nature of its hooks and by the absence of the 3rd and 4th row of vestigeal hooks on the head; its rugose hooks also distinguish it from the former species.

Specific diagnosis.—Slender cestodes reaching a length of 5·5 mm. in alcohol preserved material and normally carrying 4 or 5 segments, of which the mature segment is always the third from the last. Rostellum carries 32 to 46 hooks in two rows; larger hooks 0·037 to 0·042 mm. long, and smaller hooks 0·028 to 0·035 mm. long. Handle and guard of large hooks markedly rugose. 28 to 46 testes in each mature segment of which those in the front half of the segment are smaller and only mature later than those in the posterior half of the segment. Uterus with 15 to 20 sacculations on either side; Onchospheres finely pitted, 0·038 to 0·039 mm. long by 0·031 to 0·034 mm. broad.

Host: Leo leo krugeri. Rbts.

Habitat: Small intestine.

Locality: Northern Transvaal.

Types in the Onderstepoort Helminthological collection.

Fam. Davaineidae Fuhrm., 1907.

Raillietina (Raillietina) pintneri (Klaptocz, 1908).

This species was well represented in all the collections, and it, together with *Octopetalum longicirrosa* Baer, appears to be the commonest cestodes of Guinea fowls. The longest specimens attained a length of 76 mm. In the writer's material there were from 200 to 220 rostellar hooks which were from 0·0084 to 0·0095 mm. long; these figures are slightly in excess of those of Baer (1925), who also gives the findings of Klaptocz, and Fuhrmann, but in view of the similarity in the internal organisation of their material and the writer's, there does not appear to be any valid ground for doubting the identity of these materials.

Host: Numida mitrata. Pall.

Location: Small intestine.

Locality: Swaziland, Transvaal and Eastern Cape Province.

Fam. DILEPIDIDAE Fuhrmann, 1907.

Octopetalum longicirrosa Baer, 1925.

This species was also present in all the guinea fowl materials submitted to the writer for identification. Baer (1925) observed that in the last segments eggs were entering the paruterine organ; the writer did not find any eggs in the paruterine organs of his specimens.

Host: Numida mitrata. Pall.

Location: Small intestine.

Locality: Swaziland, Transvaal and Eastern Cape Province.

Joyeuxia fuhrmanni (Baer, 1924).

This species, of which about 35 specimens were collected from a genet, varied in length from 5 to 15 mm. and only those specimens from 12 to 15 mm. long had mature segments, which attained a maximum length of 2·5 mm. The rostellum was about 0·095 mm. long and about 0·055 mm. broad at its base and carried 14 to 16 rows of rose-thorn hooks, each about 0·0055 mm. long. The head was about 0·23 mm. wide and the four suckers were slightly oval, measuring 0·08 by 0·087 mm. in diameter. The number of segments varied from 30 to 44, and the genital rudiments could just be determined from the 5th segment; the 25th or 26th segment was mature and eggs began to appear from about 10 segments lower.

The cirrus sac is elongate, about 0·13 mm. long in mature segments and 0·145 mm. long in ripe segments, with a maximum thickness of 0·04 mm.; it extends almost transversely across the excretory canals; the cirrus is smooth and when fully everted was from 0·18 to 0·19 mm. long by 0·015 to 0·017 mm. broad. The *vasa differentia* extend along the anterior margin of the segment, and those of the two sides may often meet. The testes are large, round to oval, and vary from 40 to 50 in number; they fill up the whole area in the centre of the segment between the female glands and the male ducts; none are found anterior of the *vasa differentia*. The eggs, which are lodged singly in their capsules, are thin-shelled and slightly oval, measuring 0·039 to 0·041 mm. long by 0·028 to 0·032 mm. broad; they are closely packed together and fill up the whole space between the excretory canals, and a few may even be found exterior of these canals. In the ripest segments eggs may also be present which have not yet developed a shell or hooklets.

Host: Genetta rubiginosa. Puch.

Habitat: Intestine

Locality: Northern Transvaal.

Discussion.—From the literature it appears that two species of tapeworm, which may be referred to this genus, have been described from Genets, namely *Dipylidium dongolense* Beddard, 1913, from *Genetta dongolana* and *Dipylidium gervaisii* Setti, 1895, from *G. tigrina* (= *G. abissinica*) and *G. genetta*; in addition *Dipylidium genettae* (Gervais, 1847), from *G. genetta* might also belong to the genus Joyeuxia but according to Baer (1927) its description is too incomplete for any determination. According to this author and to Wittenberg (1932), the description of Beddard's species is also too incomplete for specific comparison, and the same also applies to that of Gervais. Accordingly, the writer is limited for comparison to *J. fuhrmanni* Baer, 1924, and *J. pasqualei* (Diamare, 1893). Wittenberg is of the opinion that these two species are cospecific but in a previous communication (1933) the writer has expressed the view that they are different. The material described above strengthens the writer's view, because except for their smaller size and consequent lesser number of segments, the genitalia and their internal arrangement is practically identical to that found in the material collected by the writer from a domestic cat; the writer's genet material is unfortunately not fully extended and consequently the internal organs appear more closely packed than in his cat material. As fully half the writer's genet material does not show any ripe segments, it is probable that these specimens represent a fairly recent infection and that the worms had not yet reached their maximum length.

Fam. ANOPLOCEPHALIDAE Fuhrmann, 1907.

Anoplocephala (*S.L.*) *genettae* sp. n.

A single immature specimen was obtained from the small intestine of a Genet in association with *Joyeuxia fuhrmanni*; it was 18 mm. long and had a maximum breadth of 1·4 mm. at its posterior end. The head is dorso-ventrally flattened, 0·56 mm. broad and 0·39 mm. long, and is not provided with a rostellum; the four suckers are rounded and about 0·23 mm. in diameter. There is practically no neck, as traces of segmentation can be made out almost immediately behind the head. The segments are broader than long, the maximum length being 0·39 mm. From the stained and mounted specimen the following details could be made out.

There are about three to five longitudinal excretory canals on either side, these being united to each other by irregularly placed transverse ducts. The outermost, representing the ventral duct, is the largest. The genital pores are all unilateral in position and situated on a slight prominence in the anterior third of the segment.

The ovary is diagonally placed on the poral side of the midline; it consists of two lobed parts separated by a pyriform receptaculum seminis, at the posterior margin of which a rounded yolk gland is attached (Fig. 2). The vagina passes almost straight to the genital pore, its distal portion, however, being larger and surrounded by darkly staining cells; this distal portion has very fine spines on its internal surface. The cirrus pouch opens anterior of the vagina; it is pyriform and does not reach the excretory canals. At the posterior end of the strobila it is 0·125 mm. long by 0·058 mm. broad. The cirrus is armed with very minute spines. There are from 30 to 45 testes which do not extend laterally beyond the outermost excretory canal; there are from 7 to 12 testes on the poral side of the ovary, 1 to 3 immediately behind the ovary and 22 to 30 on the aporal side of the ovary; the largest testes measure 0·04 by 0·05 mm. The genital ducts appear to pass over the dorsal side of the excretory canals.

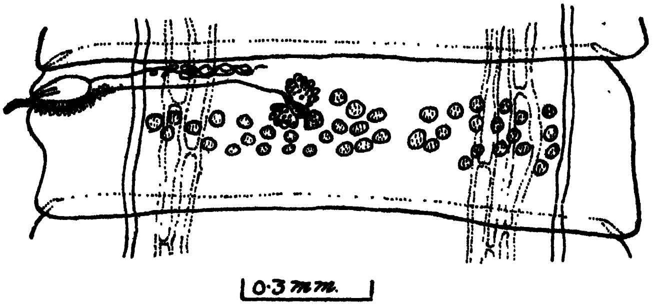


Fig. 2.—*Anoplocephala genettae* sp. n. Mature segment.

Discussion.—The absence of ripe segments showing the nature and fate of the uterus makes it difficult to assign this species to its proper systematic position among the Anoplocephalidae. However, the characters of its excretory system, and the nature and conformation of its genitalia do not fit in with any known species of this group. With access to more material it is hoped that its proper position may be determined, and in the meantime it is being placed in the genus Anoplocephala (S.L.).

Specific diagnosis.—Anoplocephalinae possessing 3 to 5 longitudinal excretory canals on either side, these canals being joined to each other by irregularly placed transverse canals. Genital pores unilateral; ovary slightly poral and two-lobed; cirrus pouch does not reach excretory canals; 30 to 45 testes extending lateral and posterior of ovary; genital ducts pass dorsal of excretory canals.

Host: Genetta rubiginosa. Puch.

Location: Small intestine.

Locality: Northern Transvaal.

Type in the Onderstepoort Helminthological Collection.

Fam. Acoleidae Fuhrmann, 1907.

Gyrocoelia kiewietti sp. nov.

Only a single specimen of this interesting cestode was obtained from the small intestine of a Blacksmith plover (*Hoplopterus armatus*) shot at Odendaal's Rust, O.F.S. The specimen was unfortunately much shrunken and no ripe egg-bearing segments were present. Sufficient data, however, were obtained to warrant the following description and for assigning it to a new species.

The entire worm was 45 mm. long and had a maximum breadth towards its posterior end of 4·5 mm. The head is slightly set off from the rest of the strobila and is 0·58 mm. across and 0·23 mm. long (Fig. 3); it carries four slightly oval and unarmed suckers measuring 0·23 by 0·26 mm. The rostellum is everted and nipple like, 0·116 mm. long by 0·104 mm. across at its base; on its outer surface it carries a single row of 84 hooks arranged in six loops; there are 14 hooks to each loop and two loops are situated on each of its dorsal and ventral surfaces and one loop on each of its lateral faces. Each hook has a relatively long handle and small guard and blade; from the tip of the blade to the tip of the handle it measures 0·029 mm. and from the tip of the blade to the tip of the guard 0·007 mm.; the blade is 0·003 mm. long and the guard 0·006 mm. long. The neck is short and broad, measuring 0·14 and 0·52 mm. respectively. The first definite segments are nearly ten times as broad as long; as the segments grow older the ratio between breadth and length decreases but even in the oldest segment these are considerably broader than long.

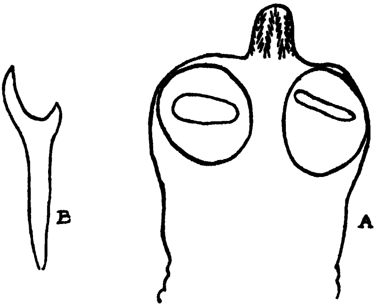


Fig. 3.—*Gyrocoelia kiewietti* sp. n. A. Scolex. B. Rostellar hook.

The genital pores alternate regularly except in a few segments where 2 or 3 genital pores are adjacent; they are situated in the centre of the lateral margin and lead into a deep genital sinus, 0·26 mm. deep. The genital ducts pass over the dorsal surface of the nerve and between the excretory canals. The cirrus sac is large, muscular and oblong and extends beyond the excretory canals; it is about 0.75 mm. long with a maximum thickness of 0·15 to 0·2 mm. (Fig. 4). The cirrus is heavily spined and occupies only the proximal half of the cirrus sac, while the remainder is filled by the much coiled and thick-walled vas deferens; in the fully extended condition the cirrus reaches a length of 0·63 mm. and has a thickness of 0·1 mm.; after emerging from the cirrus sac the vas deferens forms only a few small coils towards the anterior margin of the segment.

What may possibly be the testes is represented by a mass of follicles extending through the breadth of the segment between the cirrus sacs; these stain a dark purple with Ehrlick's acid haematoxylin; a careful search, however, did not reveal the presence of typical spermatozoa with heads and tails; in older segments these follicular cells are replaced by a granular mass consisting of isolated small nuclei. As no nucleated structures which might afford a clue were present in the male genital ducts, the writer is not able to state definitely whether this granular mass represents atypical spermatozoa or not.

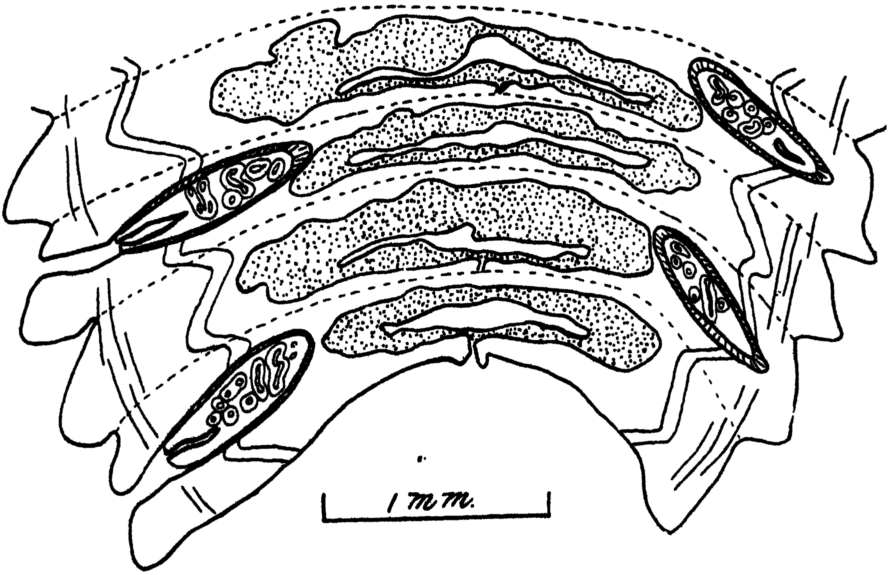


Fig. 4.—*Gyrocoelia kiewietti* sp. n. Horizontal section showing nature of uterus and cirrus sac.

The ovary is centrally placed and small and consists of relatively few tubules; it only makes its appearance after the above described testes have disappeared, i.e. in the posterior half of the strobila. As this organ appears to be immature, the writer is inclined to the view that the whole strobila had not yet attained sexual maturity, a view which is supported by the fact that even immature eggs are entirely absent. The yolk gland is represented by a few rounded cells posterior of the ovary. A vagina is entirely absent. The typical ring-shaped uterus makes its appearance very early as a transverse tube passing through the yolk gland; it continues to grow and when it reaches the excretory canals it curves forwards and then inwards again in front of the ovary to form a complete ring enclosing the ovary; its lumen enlarges and its wall becomes sacculated. A mucin-like mass, without a definite structure, occupies the greater portion of the uterus; it stains a dark purple with haematoxylin.

In the older segments there are two uterine pores to each segment, one in the centre of the posterior margin of each dorsal and ventral surface; it leads into a uterine canal which joins on to the posterior limb of the uterus. The uterine canal develops in a darker staining mass of cells extending dorsally and ventrally from the uterus to the external surface opposite.

Musculature.—The musculature is very strongly developed and the longitudinal muscles are arranged in two distinct layers, an outer consisting of a single layer of muscle bundles each composed of 30 to 50 fibres and separated by a sheet of circular muscle from the inner layer consisting of one or two layers of muscle bundles, each bundle composed of 50 to 200 fibres. A sheet of circular muscle fibres separate the inner longitudinal muscle layer from the medulla, and a thin sheet of similar muscles lie on the outer border of the outer longitudinal muscle layer. Numerous transverse muscle fibres pass irregularly dorso-ventrally through the parenchyma. The medulla has a thickness of about 0·23 mm. and the cortex 0·52 mm., of which about two-thirds is occupied by the longitudinal muscle layers.

Discussion.—Up to the present six species of cestodes have been assigned to this genus, all of which have been recovered from Charadriiform birds. In four of these, namely, *G. brevis* Fuhrmann, 1900, *G. leuce* Fuhrmann, 1900, *G. paradoxus* (v. Linstow, 1906), and *G. fausti* Shen, 1932, the number and arrangement of the rostellar hooks are known; the first two each have 40 rostellar hooks arranged in 4 loops, and the last-named has 78 hooks arranged in 6 loops; Shen's species has 66 hooks but the number of loops is not stated. The number and arrangement of the hooks in the writer's specimens easily distinguish it from Fuhrmann's two species; the arrangement, however, is similar in the writer's and von Linstow's species; the size of the hooks is also the same in both, but the number is different; the number and size of the hooks in Shen's species are different to the writer's species; a distinguishing character, however, is that in von Linstow's species there are three testes arranged in a triangle in the centre of the segment. The number of testes also distinguishes the writer's species from the two species *G. australiensis* (Johnson, 1910) and *G. perverse* Fuhrmann, 1899; the former has 50 testes according to Maplestone and Southwell (1922), these authors considering the 5 testes described by Johnston to be detached ovarian acini; the latter species has only 4 testes.

Fuhrmann (1932), in his diagnosis of this genus, decribes the rostellar hooks as "*disposés en zig-zag et formant huit angles*"; with the further knowledge we now have at our disposal, this has to be enlarged to read "*hooks arranged in a zig-zag, forming eight or twelve angles*".

Specific diagnosis.—Acoleidae with well developed rostellum carrying 84 hooks in 6 loops, two of which are dorsal, two ventral, and one on each lateral face. Genital pores, with few exceptions, alternate regularly. Testes(?) consist of a mass of follicles extending through the breadth of the segment between the inner limits of the cirrus sacs. Ovary small (? immature) and centrally placed; uterus typical of genus; eggs not known.

Host: Hoplopterus armatus. (Burch.)

Location: Small intestine.

Locality: Odendaal's Rust, Orange Free State.

Type slides in Helminthological Collection, Onderstepoort.

NEMATODA.

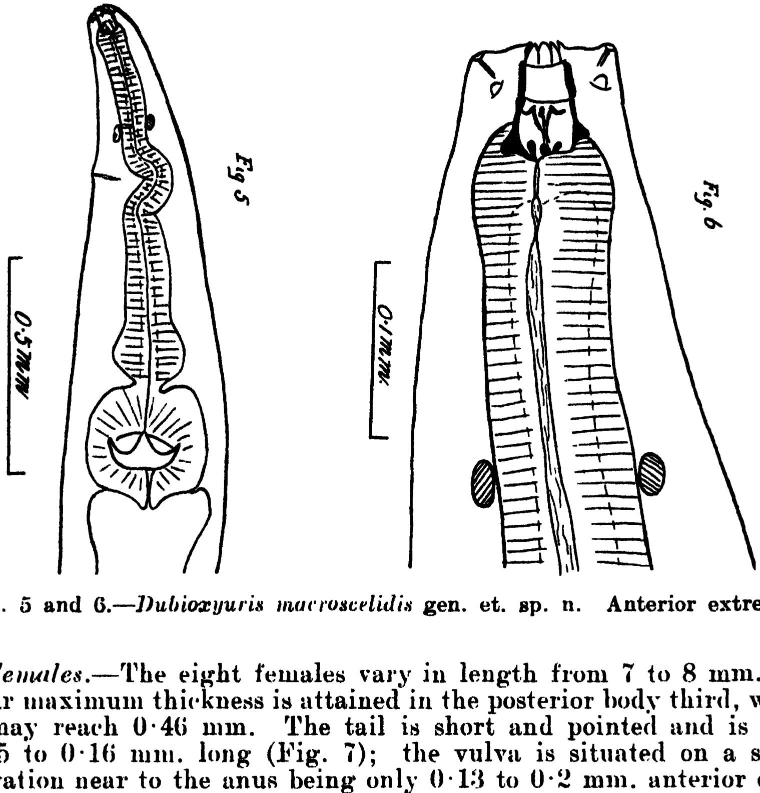
Fam. DUBIOXYURIDAE nov.

Dubioxyuris macroscelidis, gen. and sp. nov.

Six males and eight females of this interesting nematode were collected from the large intestine of an Elephant shrew. Superficially these parasites have a great resemblance to oesophagostomes except that they have a reddish colour; a cursory microscopic examination, however, quickly showed that this resemblance was only superficial, and that these parasites show a combination of characteristics not associated with any hitherto known family of nematodes. In consequence the writer cannot fit these parasites into the present scheme of classification and he proposes to create a new family of the Oxyuroidea for their reception.

The head is truncate and is not set off from the rest of the body. The cuticle is smooth and is interrupted at the anterior end by the two prominent lateral head papillae, each giving exit to a minute duct, and by the four submedian head papillae which are situated slightly behind the lateral head papillae. A pair of small wart-like cervical papillae is situated about half way down the length of the oesophagus; the excretory pore is situated 0·08 to 0·1 mm. in front of these papillae.

The mouth is round and is bordered by a ring of 11 leaf elements (*corona radiata*) (Figs. 5 and 6); it leads directly into the cylindrical buccal capsule whose diameter is equal to its depth (0·055 mm. in the female and 0·021 mm. in the male), and whose wall is thickened; this capsule leads into a second or pharyngeal capsule sunk into the anterior face of the oesophagus; the pharyngeal capsule is deeper than it is wide, being 0·038 mm. deep and 0·028 mm. wide in the male and 0·075 mm. deep and 0·06 mm. wide in the female; its wall is also thickened and externally in its posterior third it is provided with a thicker cuticular ledge; its inner surface carries three large teeth arising from the base and sides of the capsule and extending forwards almost the whole depth of the capsule; in addition two small club-like teeth are situated on either side of the base of each large tooth. The oesophagus, which is from 1·0 to 1·1 mm. long is double-bulbed, having the same shape as that found in the genus Subulura; anteriorly it is slightly thickened to form a small head into the anterior end of which the pharyngeal capsule is sunk; this portion is from 0·07 to 0·087 mm. thick; the portion immediately following measures 0·06 to 0·078 mm. in thickness; it increases gradually posteriorly to attain a maximum thickness of 0·16 to 0·19 mm. in males and females respectively in the middle of the anterior bulb; a sudden constriction joins this bulb to the one following which is broader than long, reaching a maximum of 0·26 by 0·3 mm. in the male and 0·26 by 0·32 mm. in the female.



Figs. 5 and 6.—*Dubioxyuris macroscelidis* gen. et. sp. n. Anterior extremity.

Females.—The eight females vary in length from 7 to 8 mm. and their maximum thickness is attained in the posterior body third, where it may reach 0·46 mm. The tail is short and pointed and is from 0·15 to 0·16 mm. long (Fig. 7); the vulva is situated on a slight elevation near to the anus being only 0·13 to 0·2 mm. anterior of it. At a distance of 0·15 to 0·17 mm. anterior of the vulva the body suddenly swells out and becomes very much thicker, whereas from this level to the tail tip the body tapers uniformly; the thickness of the body at this level is 0·18 to 0·2 mm. whereas immediately anterior it is from 0·3 to 0·35 mm. thick. The vagina is from 0·44 to 0·75 mm. long by 0·04 to 0·065 mm. wide (Fig. 8); it passes anteriorly and during its course may be looped; its anterior end is provided with a sphincter about 0·08 mm. long. After the sphincter it splits into two limbs, which, however, are peculiar in that they are dissimilar; the left limb, which may be regarded as a utero-duct, is provided with a sphincter about 0·04 mm. long soon after its origin; it passes forwards for a distance of 0·5 to 0·75 mm. after which it bends back again to about the level of its sphincter when it again bends forwards to join the uterus proper at about the level of its most anterior bend; this convoluted tube maintains a more or less uniform thickness of about 0·045 mm. in young specimens but in mature specimens it is filled with eggs which consequently distend it. The uterus itself is a fusiform sac passing straight forwards and contains eggs in different stages of development; the right limb or utero-duct is similar to that of the left except that its first portion is very much enlarged and it is not provided with a sphincter near its junction with the vagina. What the function of this sac is, is not clear as it did not contain a single egg in any of the females, notwithstanding that the remaining portions of the utero-duct and the uterus contained abundant fully developed eggs; consequently it cannot be regarded as an egg-reservoir; all these sacs were, however, filled with a granular material, some of which even passed for some distance up the utero-ducts; it is possible that this granular material represents stored spermatozoa, in which case these sacs would be receptacula semini.

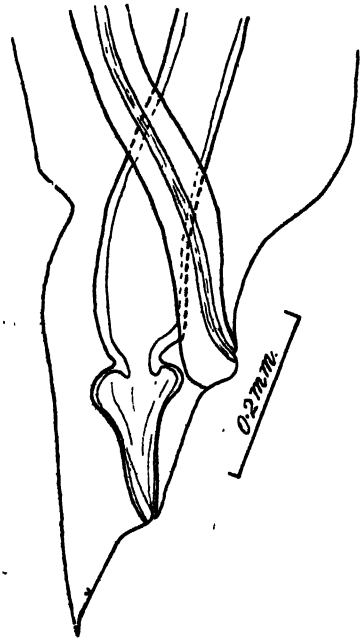


Fig. 7.

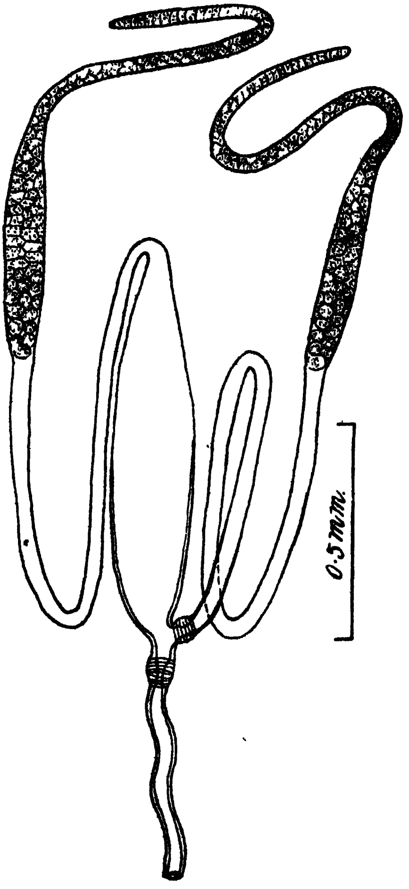


Fig. 8.

Fig. 7.—*Dubioxyuris macroscelidis* gen. et. sp. n. Posterior extremity of female.

Fig. 8.—*Dubioxyuris macroscelidis* gen. et. sp. n. Female genitalia of young specimen.

The eggs are rounded to oval and are embryonated *in utero*; they have a thickened shell and their size varies from 0·055 to 0·58 mm. in length by 0·046 to 0·048 mm. in breadth.

Males.—The six males vary in length from 6 to 7 mm. with a maximum thickness of 0·34 mm. in the middle of the body. The caudal extremity (Figs. 9 and 10) is provided with an ample membrane, supported by eight pairs of papillae, and the tail; the membrane extends across the ventral surface from the tips of the first or most anterior papilla of either side; the last 0·025 mm. of the tail is free; on each side of the anterior half of the tail there are three small rays passing almost to the edge of the bursa; the remaining five rays of either side support the lateral lobes of the caudal membrane; all extend to the edge of the membrane and the anterior three are the largest, the posterior two being much smaller; the central ray is the longest.

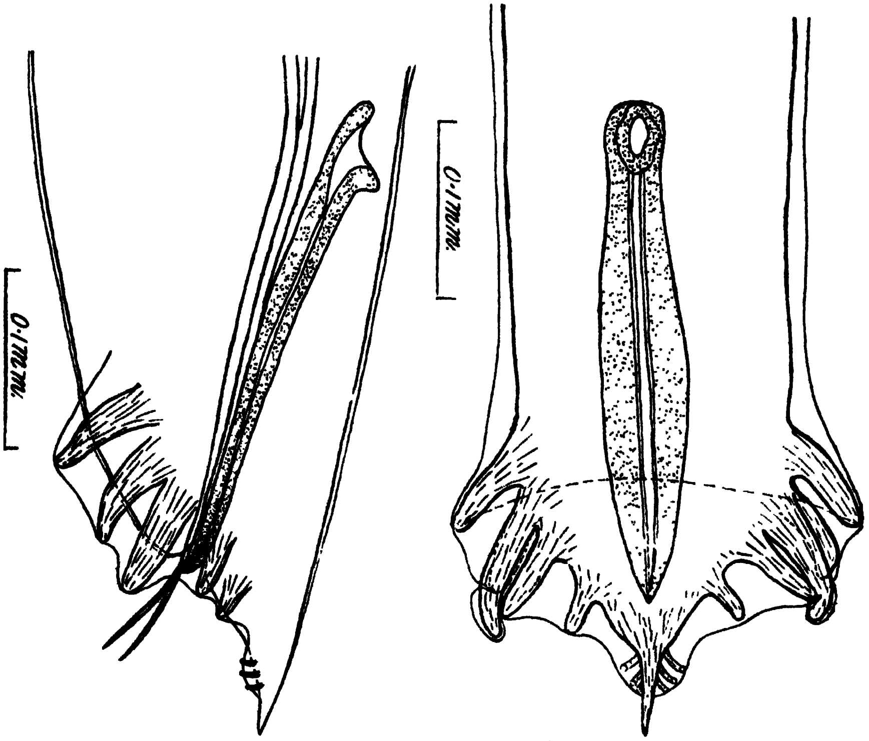


Fig. 9. Fig. 10.

Figs. 9 and 10.—*Dubioxyuris macroscelidis* gen. et. sp. n. Lateral and dorsal view of male posterior extremity.

The two pairs of equal and similar spicules are relatively long, varying in length from 1·6 to 1·75 mm.; they have a thickness of 0·02 mm. at their base after which they taper gradually to terminate in fine points. The gubernaculum is large and massive and may protrude through the cloaca; in a dorsal or ventral view it is somewhat dagger-shaped with a midrib and lateral flanges; its maximum width in its middle is 0·046 mm. of which 0·017 mm. on either side of the midrib represents the flanges. In lateral view it is seen to be provided with an anterior head tilted dorsalwards and from here it tapers backwards to end in a bluntly rounded tip tilted ventralwards. Its total length varies from 0·275 to 0·296 mm.

Affinities.—The presence of a mouth capsule with a corona radiata, the presence of what might be considered to represent a caudal bursa with atypical ray formation, and the nature of the female caudal extremity would suggest that this parasite had some affinities with the Strongyloidea. Against this we have the definite two-bulbed nature of the oesophagus and the nature of the female genitalia suggesting Oxyuroid relationships. In the former case the characters concerned are external, whereas in the latter internal characters are concerned; the writer believes that in determining relationships internal characters, which are not so liable to change to suit different conditions, are a better guide, and are to be given a greater importance than external characters. Applying this rule the writer feels that these parasites must be considered as belonging to the Oxyuroidea. In this superfamily we find forms having a similar oesophagus and comparable mouth structures in the Subulurinae; in the Oxyurinae, especially in some forms from tortoises, we find the lips broken up to form structures which may be regarded as the forerunners of a corona radiata; caudal membranes leaving a portion of the tail free are also present in this suborder and these membranes are supported by papillae which are not arranged on a definite plan as is the case with the rays of the Strongyloidea. In addition the female genitalia of the Oxyuroidea show far more extremes of variation than those of the Strongyloidea.

Among the Oxyuroidea the writer feels that these parasites may be placed near the family Oxyuridae with whose members it agrees in having a bulbed oesophagus, double female genitalia with special modification and no special development of the ventral precloacal muscles in the male; but the first two characters (except modification of genitalia) together with the gross nature of the oesophagus with its pharyngeal capsule and teeth also suggests relationships with the Subuluridae notwithstanding the absence of specially developed precloacal muscles. The writer feels that these parasites occupy an intermediate position between these two sub-families and that a special family—DUBIOXYURIDAE—must be created for their reception; this family would be characterised by the presence of a corona radiata, buccal and pharyngeal capsules, an oesophagus with a double bulb, two uteri, well-developed caudal expansions in the male, two spicules and a well-developed gubernaculum.

Specific Diagnosis.—Oxyuroidea without lips but carrying a corona radiata round mouth; mouth and pharyngeal capsules present, the latter provided with three large and twelve small club-like teeth; oesophagus with two bulbs; vulva near to anus; tail short and pointed; right utero-duct very much enlarged and sac-like; caudal alae of male well developed and supported on either side by 5 large papillae and 3 small papillae on the tail; two spicules equal and similar and relatively long; gubernaculum large, massive and somewhat dagger-shaped in dorsal or ventral view.

Host: Macroscelides proboscideus. (Shaw.)

Location: Large intestine.

Locality: Western Transvaal.

Types in the Onderstepoort Helminthological collection.

Fam. HETERAKIDAE Raill. and Henry, 1914.

Subulura suctoria (Molin, 1860).

Of the species of Subulura from gallinaceous birds the three species *S. suctoria* (Molin, 1860), *S. differens* (Sonsino, 1890) and *S. brumpti* (Lopez Neyra, 1922) would appear from the literature to be very closely related; the writer has unfortunately not been able to consult the original descriptions, and has had to rely on the redescriptions compiled by Cram (1927). From Cram's paper it appears that the first-named species may be distinguished from the other two species in that it possesses 11 pairs of caudal papillae in the male, and in that it is larger and has lateral alae which extend only half the length of the oesophagus. *S. differens* and *S. brumpti* are distinguished from each other in that in the latter the male tail is longer, the gubernaculum may be twice as long as in the former species and the eggs are very large.

With regard to the papillae on the tail of *S. suctoria* the writer doubts whether the statement that there are 11 pairs is correct: judging from the description and figure given by Cram the writer thinks that the caudal pores have been mistaken for papillae. Further, the writer does not think that the size and nature of the alae can in this case be employed as specific differences, because after examining numerous specimens obtained from domestic and guinea fowls fixed in alcohol or formalin, the writer found that the size of the adults varied considerably, being from 10 to 19 mm. long for the females; if the worms were allowed to die in cold water the worms stretch enormously, and females have been found to reach 25 mm. in length; as to the alae the writer also noted considerable variations and they were found to be from about half the length of the oesophagus to about half again as long as this organ; usually the alae extended to just posterior of the oesophagus. From these remarks it is clear that the supposed differences between *S. suctoria* on the one hand and *S. differens* and *S. brumpti* on the other are not valid.

The close relationship of these three species is also shown by their ovejectors, which are built on the same plan consisting of a pyriform and muscular vestibule, a saccular sphincter terminated by a thickened glandular portion, and a terminal " tromp ". This type of ovejector is also present in the writer's materials from domestic and guinea fowls. The writer dissected out about two dozen ovejectors from materials obtained from both hosts, and he found that variations were also present in the sizes of the different parts of this organ; however, it was noted that in all cases the sphincter was about half the length of the vestibule; a constant feature, however, was that in all cases the sphincter joined the vestibule laterally on its dorsal side, and a conspicuous plug protruded into the vestibule at this point.

From Cram's data the sphincter has the same length as the vestibule in *S. differens*, about five-sixths of the vestibule length in *S. suctoria* and nearly two-thirds of the vestibule length in *S. brumpti* and in addition the total length of the ovejector is about 1 mm. in this last species. Whether these data have been obtained from the examination of one or several specimens of each species it is unfortunately not clear, but considerable variations can be expected in the size of these muscular structures, depending on the method of killing and fixation of the material, and consequently the writer is not inclined to attach any specific significance to the difference between these data and the writer's observations.

The writer is not inclined to accept the supposed specific difference in the length of the tail and size of the egg in *S. differens* and *S. brumpti;* considerable variations were noted by the writer in his materials, variations extending over the sizes given for these two species; in preserved material this was especially the case with the eggs which, due to their thin shells, were liable to undergo considerable deformations.

In the writer's material the only striking difference between the materials from domestic fowls and guinea fowls was the length of the spicule; in the former the spicular length never exceed 1·2 mm. whereas in the latter the lengths were from 1·2 to 1·6 mm. According to Cram's data the first-named materials would correspond to *S. suctoria* and *S. differens* and the other materials to *S. brumpti*. However, apart from these spicular variations, the writer's materials are so similar in all other respects (absence of lips, shape and depth of buccal capsule, alae, ovejectors, etc.), that he cannot but regard all his material as being cospecific.

From the literature these three species all have no lips or they are stated to be indefinite; they possess lateral alae; the head is bent dorsally; three small teeth are present at the anterior end of the oesophagus; they have equal spicules which are of the same shape, pointed and from 1 to 1·5 mm. long; and the ovejectors are of the same type. In addition the writer maintains that they also all have 10 pairs of caudal papillae in the male. These characters, supported by the presence of similar characters in the writer's materials, make him conclude that these species are cospecific, and, Molin's name having priority, the correct name is *Subulura suctoria*.

Subulura dentigera sp. nov.

This species occurred in guinea fowls, in association with the preceding species, and was far more abundant; all the guinea fowls examined harboured this species, and as this host originated from Swaziland, Northern Transvaal, Potchefstroom and Lady Grey, it is seen that this parasite has a very wide distributioin in South Africa.

This parasite has an external appearance typical of the members of this genus. The anterior end is bent dorsalwards and carries two prominent lateral alae generally extending to just behind the end of the oesophagus. The mouth, however, is bounded by two distinct and hemispherical lateral lips each carrying on its outer surface a median lateral papilla and two submedian papillae (Fig. 11, B and C); internally its cuticular lining is thickened to form a row of 5 or 6 denticles, 0·003 to 0·004 mm. high, and running parallel to the anterior margin of the lip; the presence of these denticles is, as far as the writer has been able to ascertain, the first record of these structures occurring in any member of the Subulurinae. The mouth is a dorso-ventrally elongated oval aperture and leads into a dorso-ventrally elongated buccal cavity whose wall consists of thickened cuticular material; it is from 0·028 to 0·04 mm. long by 0·014 to 0·019 mm. wide, and its depth from 0·015 to 0·02 mm. in the males and 0·02 to 0·023 mm. in the females; thus the depth of the mouth capsule is considerably less in this species than in *S. suctoria* and the section of its wall is also quite different (Fig. 11 A). There are three minute teeth at the anterior end of the oesophagus, similar to those found in *S. suctoria.*

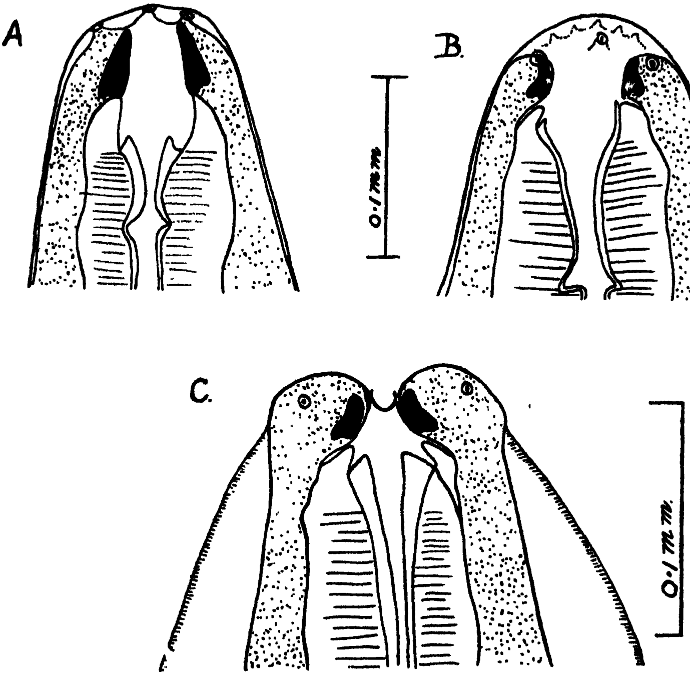


Fig. 11A.—*Subulura suctoria.* Lateral view of head.

Figs. 11B and 11C.—*Subulura dentigera* sp. n. Lateral and ventral view of head.

The females are 13 to 22 mm. long and the body has the same external appearance as in the male, except that the tail is straight and pointed; their maximum thickness across their middle is from 0·46 to 0·58 mm. The oesophagus is from 1·07 to 1·35 mm. long, and the nerve ring and excretory pore are found about 0·3 and 0·42 mm. from the anterior end respectively. The tail, which is long and is terminated by a spike-like tip, is from 0·84 to 1·05 mm. long. The vulva is non-protuberant and situated in the anterior body half; its position divides the body into the ratio of 2:3 to 3:4. The ovejector, which is from 0·43 to 0·64 mm. long, passes forwards and inwards and is built on the same plan as that of *S. suctoria*, except that the sphincter enters the vestibule at its anterior dorsal corner and not quite so lateral as in *S. suctoria* (Fig. 12); the vestibule is from 0·38 to 0·46 mm. long, the sphincter including its glandular portion from 0·15 to 0·23 mm. long, and the tromp from 0·75 to about 1 mm. long. The eggs are oval, thin shelled and embryonated *in utero*. They are 0·07 to 0·081 mm. long by 0·058 to 0·061 mm. broad.

The males are 9 to 10 mm. long with a maximum thickness of 0·39 to 0·45 mm. across the middle of the body. Attenuation of the body anteriorly is more marked than posteriorly; across the base of the lips the body thickness is only 0·07 to 0·075 mm. The oesophagus, 1 to 1·1 mm. long, is of the usual shape and it is encircled by the nerve ring about 0·24 mm. from the anterior end of the body. The excretory pore is situated about 0·15 mm. posterior of the nerve ring. The posterior extremity is strongly arched ventralwards and the tail is provided with two distinct alae, 0·02 to 0·24 mm. wide; the tail, which is from 0·22 to 0·26 mm. long is terminated by a spike-like portion about 0·06 mm. long. There are 10 pairs of caudal papillae, arranged exactly as in *S. suctoria*, i.e. three pairs preanal of which two pairs are situated lateral of the sucker, two pairs adanal, and five pairs postanal; the minute caudal pores are found just posterior of the 3rd postanal pair. The spicules are of equal length, and alate and terminate in sharp points; they vary in length from 1·3 to 1·5 mm. with a thickness of 0·023 to 0·026 mm. about 0·1 mm. behind their proximal or head ends. A well chitinized gubernaculum is present, which is from 0·15 to 0·165 mm. long; in side view it may be straight or slightly arched, and its proximal third appears thickened; in dorsal view it is seen to be somewhat Y-shaped (Fig. 13).

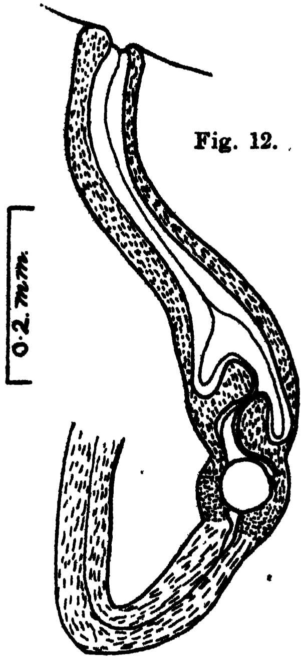


Fig. 12.

Fig. 13.

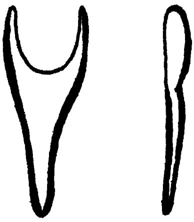


Fig. 12.—*Subulura dentigera* sp. n. Vagina.

Fig. 13.—*Subulura dentigera* sp. n. Dorsal and lateral view of gubernaculum.

Affinities.—The nature and length of the spicules, arrangement and number of the caudal papillae in the male, and the structure of the ovijector, ally this species to *S. suctoria* (Molin, 1860). It may, however, be easily distinguished from this species by its two lips each carrying a dentigerous border, the shape and depth of the buccal capsule and in that the sphincter joins the vestibule somewhat anteriorly.

Specific diagnosis.—Subulurinae reaching 10 mm. in length in the male and 22 mm. in the female, provided with two distinct lateral lips, having an internal dentigerous border. Buccal capsule shallow and dorso-ventrally elongated; three minute teeth at its base; lateral alae extend to anterior end of intestine. Spicules equal, pointed, 1·3 to 1·5 mm. long; gubernaculum Y-shaped; 10 pairs of caudal papillae; vulva in anterior body half; ovejector of vestibule, sphincter and tromp; sphincter enters vestibule at its antero-dorsal corner.

Host: Numida mitrata. Pall.

Situation: Caeca.

Locality: South Africa.

Type in the Helminthological collection at Onderstepoort.

Heteroxynema vlakhaasi sp. n.

This species is represented by one male and eight females from a hare shot in the Potchefstroom district and one male and one female from the same species of hare shot in the Klerksdorp district. Outwardly the specimens are very similar to the *Subulura suctoria*. The anterior end is in all cases bent dorsalwards to form a hook; the posterior extremity of the female is straight but that of the male is hooked ventralwards.

There are two well-developed lateral cervical alae 0·09 mm. broad and extending from immediately behind the lips to about 0·5 mm. posterior of the oesophagus (Fig. 14). These alae are hyaline and show no transverse markings; cervical papillae are absent. The mouth is provided with a dorsal and two sub-ventral lips, each somewhat dome-shaped and each carrying two papillae. The mouth cavity is very small and at its base there are three very small pointed teeth situated one on the anterior face of each of the three oesophageal segments.

The oesophagus is club-shaped and relatively short, being just over 1 mm. in the male and from 1·5 to 1·6 mm. long in the female; it is provided with a muscular bulb at its posterior end, but this bulb is not sharply constricted off from the rest of the osophagus. The bulb is 0·35 mm. long by 0·225 mm. broad in the male and 0·416 mm. long by 0·26 to 0·3 mm. broad in the female. The anterior end of the oesophagus is also slightly thicker than the rest of the oesophagus. The nerve ring is just behind this anterior thickening 0·27 to 0·28 mm. from the anterior end. An excretory pore was not observed.

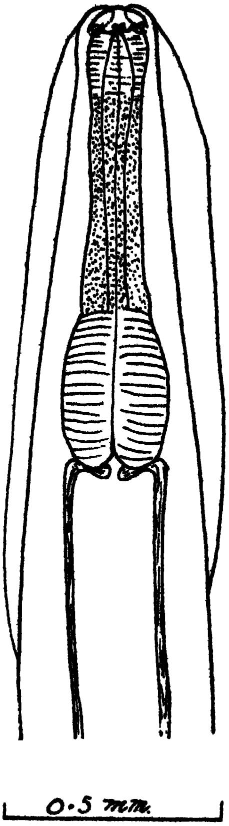


Fig 14

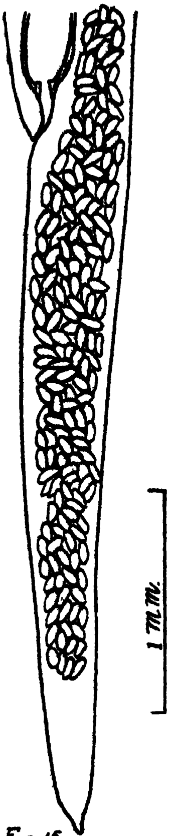


Fig 15

Figs. 14 and 15.—*Heteroxynema vlakhaasi* sp. n. Fig. 14. Anterior extremity. Fig. 15. Posterior extremity of female.

The *females* are 19·5 to 22 mm. long with a maximum thickness of 0·68 to 0·82 mm. over the middle third of the body. The tail (Fig. 15) is relatively long, 3·1 to 3·23 mm. long; it tapers gradually until near its end, when it narrows suddenly to end in a conical tip. The vulva is in the anterior body half at about the junction of the 1st and 2nd body thirds, being 6·7 to 7·4 mm. from the anterior end; it leads into a muscular vagina which passes obliquely inwards and forward (Fig. 16); it is 1·3 mm. long by 0·25 mm. thick; at its anterior end it is much thickened and then bends abruptly backwards to join an oblong swelling 0·28 mm. long by 0·17 mm. thick, 0·9 mm. from the bend. Beyond the swelling it narrows down to about 0·08 mm. and passes straight back for 2·6 mm. to join the common uterus filled with eggs; this common uterus passes further down the body and near the anus it joins the two uteri which extend down into the tail and then bending back passes forwards again to the middle body third. Numerous eggs are present and they are large and smooth-shelled and are morulated *in utero*; one side is flattened as seen in oxyurid eggs, and opposite the flattened side, more towards one end, there is an irregularly round micropyle (Fig. 17). They vary in length from 0·122 to 0·139 mm. by 0·061 to 0·072 mm. thick.

Fig. 16.

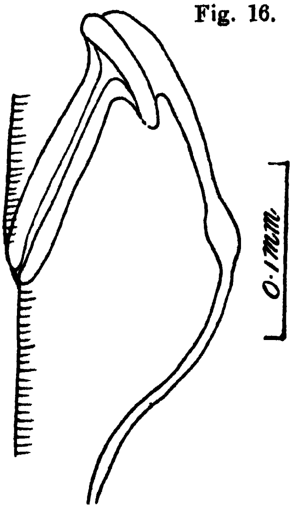


Fig. 17.

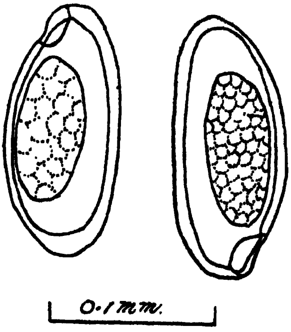


Fig. 16.—*Heteroxynema vlakhaasi* sp. n. Vagina.
Fig. 17.—*Heteroxynema vlakhaasi* sp. n. Eggs.

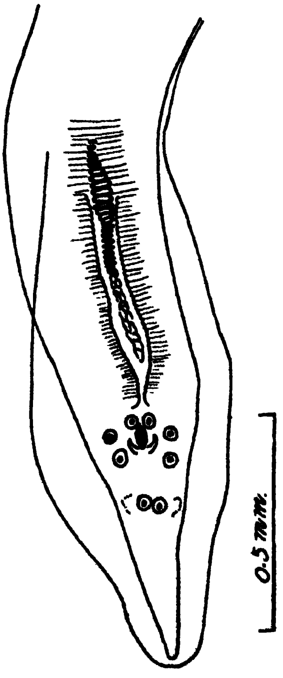


Fig. 18.—*Heteroxynema vlakhaasi* sp. n. Ventral view of posterior extremity of male.

Of the two *males* one is much shrivelled; the other is 12·5 mm. long with a maximum thickness of 0·45 mm. Two well-developed caudal alae are present which meet round the tip of the tail which is 0·484 mm. long (Fig. 18); the alae are 1·17 mm. long and attain a maximum width of 0·112 mm. There are 5 pairs of sessile papillae, four pairs of which are clustered round the cloaca; the fifth pair is ventrally placed at about the junction of the first and second tail thirds. The first pair is precloacal and ventral and immediately in front of the cloaca; the second pair is more lateral and situated at about the level of the anterior margin of the cloaca; the third pair is somewhat rose thorn-like and situated at the side of the posterior third of the cloaca; the fourth pair is more lateral and slightly posterior of the third pair. On either side of the fifth pair there is a slight cuticular elevation which, unless carefully examined, may be misinterpreted as an additional pair of caudal papillae.

In front of the 1st caudal papillae there is an elongate cuticular depression 0·4 mm. long; this is bounded on either side by a cuticular flange formed from the body cuticular lining and having the cuticular body annulations more strongly developed; these flanges do not met anterior and posterior of the depression. In the depression itself there is a streak of cuticular elevations which increase in size anteriorly and at the anterior limit of the depression become much enlarged and project prominently beyond the general body level; these enlarged cuticular elevations possibly play a rôle during copulation, because a dark-brown cement-like material was adhering to them, and a similar substance was also present round the vulva. Spicules and gubernaculum appear to be entirely absent, as a careful search for these structures in both specimens did not reveal any trace of their presence.

Affinities.—The presence of a bulbed oesophagus and a sucker-like depression in the male places this species among the *Subulurinae Travassos*, 1914. Of the members of this subfamily the nature of the sucker more closely resembles that of the genus *Heteroxynema* Hall, 1916, although the cuticular ornamentation described for *H. cucullatum* Hall, 1916, is not present in the species described above. However, the presence of three lips, the absence of spicules and gubernaculum, the nature of the female genitalia, and the oxyurid-like shape of the eggs definitely show that these species are closely related and co-generic. The writer's specimens, however, differ from Hall's species in their much larger size, in the presence of well-developed caudal alae in the male, and in the number and arrangement of the male caudal papillae. The larger size of both sexes and the absence of an unpaired postcloacal papilla in the male easily distinguishes the writer's species from *H. wernecki* Freitas and Almeida, 1936.

The writer has unfortunately not been able to consult a description of *H. muris* Vaz and Pereira, 1934, but according to de Freitas and de Almeida (1936) the genitalia of the female of this species differ considerably from those found in their's and Hall's species and they think that when the male is discovered this species will probably have to be placed in another genus.

Specific diagnosis.—Subulurinae provided with 3 lips, well-developed cervical alae and caudal alae in the males; three minute teeth at base of mouth cavity; oesophagus with posterior bulb; vulva in anterior body half; vagina long, eggs large, elongate and flattened on one side and provided with a micropyle; uterus extending some distance down tail; spicules and gubernaculum absent; 5 pairs of sessile caudal papillae in male, of which 4 pairs are circumcloacal and one pair at junction of 1st and 2nd tail thirds. Ventral sucker with internal longitudinal ridge carrying cuticular elevations which become large and prominent immediately anterior of sucker. Parasite of Leporidae (Hares).

Host: Lepus capensis capensis L. (Vlakhaas.)

Location: Large intestine.

Locality: Western Transvaal.

Types in the Onderstepoort Helminthological Collection.

Fam. FILARIIDAE (Cobbold, 1864) Claus, 1885.

Hyracofilaria hyracis gen. and sp. nov.

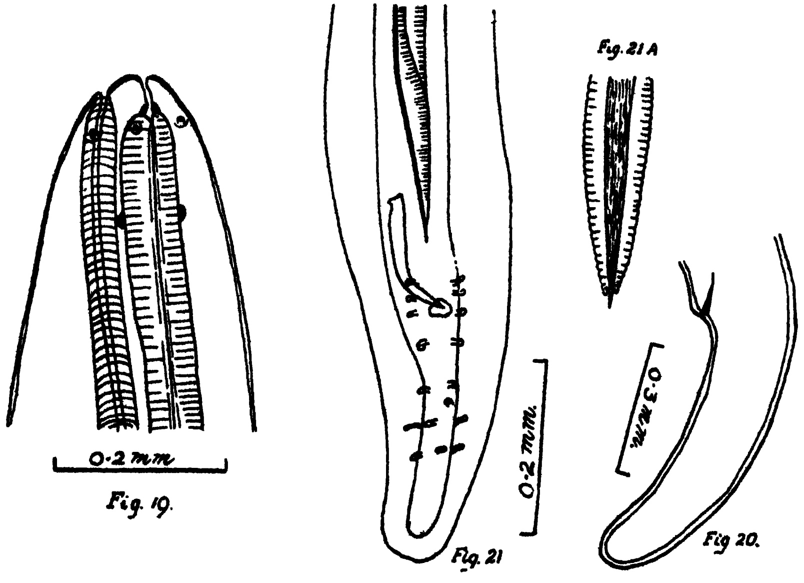
This species was represented by several complete male and female specimens, collected by Mr. Frean, Veterinary Surgeon at Potchefstroom, Transvaal; he obtained them from under the superficial muscles and fascia around the pectoral, abdominal and pudic areas of a *Hyrax* sp. shot in the Graaff-Reinet District of the Cape Province. Superficially the worms are very similar to *Setaria equina* of the horse, the females, however, being longer and thinner.

The body in both sexes has a fairly uniform thickness except towards the anterior and posterior ends, where it becomes attenuated; the anterior end is straight, but the posterior end in the female is slightly arched ventralwards and in the male forms a few loose spirals. Externally the cuticle shows no transverse annulations.

The mouth is a small rounded aperture situated in the centre of the anterior face; there are no lips, but the mouth is surrounded by a weakly-cuticularised pad whose inner surface, where it rests on the oesophagus, is strengthened by a cuticular ring (Fig. 19). Externally at the base of this pad there are the usual two lateral and four submedian papillae; a small duct traverses each lateral papilla.

The oesophagus is long, being about 6 m.m. long in the male and 12 m.m. long in the female; it consists of a short muscular portion and a much longer glandular portion. In the male this muscular portion is only about 0·12 mm. long by 0·023 mm. thick and in the female 0·45 mm. long by 0·1 to 0·17 thick; the glandular oesophagus increases in thickness posteriorly and in the male may be 0·16 mm. thick at its posterior end and in the female 0·2 mm. Cervical papillae were not seen, neither was the excretory pore seen. The nerve ring encircled the muscular oesophagus 0·1 to 0·13 mm. from the anterior end.

The *females* varied in length from 235 mm. to 290 mm., with a maximum thickness of 0·46 to 0·5 mm.; the tail (Fig. 20) is arched ventralwards, ends bluntly, and is from 0·6 to 0·7 mm. long. It carries no papillae or processes. Over the anus the body thickness has decreased to about 0·2 mm. and at the tail tip the thickness is 0·1 mm. The vulva is a ventral transverse aperture situated on the anterior face about 0·05 mm. from the mouth; it leads into a long and straight muscular vagina, 6 to 8 mm. long and about 0·12 mm. thick; this in turn divides to join the two uteri which in their initial stages pass straight down the body; only in the posterior body third do they form a few loose curves which are continued backwards to the level of the anus. The eggs are oval and thick-shelled and are embryonated *in utero*; they are from 0·05 to 0·056 mm. long by 0·035 to 0·031 mm. broad.



Figs. 19, 20 and 21.—*Hyracofilaria lyracis* gen. and sp. nov.

Fig. 19.—Anterior extremity of female.

Fig. 20.—Posterior extremity of female.

Fig. 21.—Posterior extremity of male, ventral view.

Fig. 21A.—Ventral view of tip of left spicule.

In the *male* the tail is from 0·23 to 0·26 mm. long and is loosely coiled (Fig. 20); on either side, from about 1 mm. anterior of the cloaca, an ala passes down the body and tail and round the tip of the tail; its maximum breadth of 0·06 mm. is attained anterior of the cloaca after which it narrows posteriorly to 0·02 mm. at the tip of the tail; it is provided up to the last post cloacal caudal papillae with fine transverse markings. The number and arrangement of the caudal papillae are slightly irregular, but the following, however, are constant; two pairs papillae immediately precloacal, one pair adcloacal and four pairs post coacal, on the anterior two-thirds of the tail and somewhat equidistant from each other; in addition, there are present two or three additional ventral papillae between the last three pairs of caudal papillae. The spicules are markedly unequal and dissimilar; the left is 0·64 to 0·71 mm. long and the right 0·145 to 0·152 mm.; the former is 0·11 mm. thick just behind its head and the latter 0·13 mm. The left spicule consists of two parts, an anterior portion 0·29 to 0·32 mm. long and strongly cuticularised and a posterior membranous portion; this latter portion has two lateral membranes provided with transverse marking and extending almost to the tip of the spicule (Fig. 21A). The right spicule is well cuticularised, and is slightly arched and tapers gradually to end in a rounded tip. A gubernaculum is absent.

Affinities.—The presence of a rudimentary peribuccal ring, the shape and inequality of the spicules and the anterior vulva places this species in the family *Setariinae.* Two species of the genus, *S. loveridgei* Sandground, 1928, and *S. hyracis* Baylis, 1932, have been described from hyracoids but the writer's specimens differ from these species and also from all the known genera of this subfamily in the presence of caudal alae and the absence of epaulette-like structures at the anterior end. For this reason a new genus—*Hyracofilaria*—has been created for its reception with the following diagnosis: Setariinae having a smooth cuticle, a vestigeal peribuccal ring and no lips; vulva situated very near to the mouth; vagina long and muscular; two uteri; tail of male alate; spicules very unequal and dissimilar. Type *H. hyracis* sp. nov. from *Hyrax* sp. Cape Province.

Specific diagnosis: As for genus.

Host: *Hyrax* sp.

Location: Under muscles and fascia.

Locality: Graaff-Reinet, Cape Province.

Types in the Helminthological Collection, Onderstepoort.

SUMMARY.

Seven new species of helminths are described, namely: *Echinococcus felidis* from a lion; *Anoplocephala (S.L.) genettae* from a genet; *Gyrocoela kiewietti* from a plover; *Dubioxyuris macroscelidis* from an elephant shrew; *Subulura dentigera* from a guinea fowl; *Heteroxynema vlakhaasi* from a hare; and *Hyracofilaria hyracis* from a hyrax. In addition, a new family of the Oxyuroidea, namely *Dubioxyuridae* is created for the reception of the parasite from the shrew. The status of the three species *Subulura suctoria* (Molin), *Subulura differens* (Sonsino), and *Subulura brumpti* (Nopez-Neyra) is discussed and evidence is brought forward to show that these three species are co-specific.

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Cysticercosis in Swine and Bovines, with special reference to South African conditions.*

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PART I.

Introduction.

IN discussing the etymology and history of, what we understand in the ordinary English of the meat industry, as " measles " in pork and beef, it is quite insufficient to consider the " bladderworm " stage of the parasite only, without discussing the historical advancement of knowledge regarding the adult tapeworm which succeeds the " bladderworm ". The measle, bladderworm or larva, found in the muscles of the pig, for example, and the adult resultant tapeworm of man are so closely related, that one cannot be successfully described or discussed, without investigation of the corresponding stage in the other.

It is obvious, however, that in an article on " Cysticercosis ", the bladderworm stage of the common parasite should receive the fuller consideration, although the adult, parent or final stage of the parasite must also receive attention, since we are merely dealing with two stages of a parasite, usually passed in two different hosts, and in order to arrive at a plan of campaign towards eradication of the parasite at either stage, we should understand the histology, pathological anatomy and peculiarities of both. It will be noticed that the word " usually " has expressly been used in the previous statement. The normal intermediate host of one of the two parasites to be discussed is the pig, and the final host, or host of the mature tapeworm is man. Under certain circumstances, however, man may actually be the host at both stages, and thus play the rôle, in the intermediate stage, of the pig, and be the harbourer of the pig measle. Then again, in addition to the pig and man, the dog and the monkey may also be the intermediate hosts of the pig measle bladderworm. We shall, therefore, not depart from the limits justified by the title of our subject, if we deal with tapeworm larvae of the same species as those of the pig in, for example, man, the dog, and the monkey, since the pig must in every case be considered as the original host of that particular bladderworm, which is followed by an adult tapeworm stage in man, and followed in turn by a second embryonic generation in, say, another pig, which may then be the precursor of various series of adult and embryonic generations alternating between man, the dog, man, the pig, man, the monkey, etc.

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* A Thesis approved by the University of South Africa for the Degree of Doctor of Veterinary Science.

Etymological Discussion

The intermediate, or bladderworm stage of certain tapeworms is known as the *cysticercus* stage. The pathological infection of an animal body with *cysticerci* is known as cysticercosis.

The name *cysticercus* is derived from the Greek words *Kystis*, a bladder, and *Kerkos*, a tail. (Italic, instead of Greek lettering will be used throughout this work, when names of Greek derivation are defined). The pig measle is known as *Cysticercus cellulosae*, the bladderworm of the cellular or connective tissue, on account of its usual location in the connective tissues between fibres. The beef measle is known as the *Cysticercus bovis*, i.e., the bladderworm of the ox, or in some parts of Europe it is called *Cysticercus inermis*, the unarmed bladderworm.

The adult tapeworms of the above-mentioned *cysticerci* are the *Taenia solium* and the *Taenia saginata*, respectively. The Latin name *Taenia* is derived from the Greek word *Tainia*, a ribbon or a fillet. *Taenia solium* means the solitary tapeworm (French " *Ver solitaire* "), a name to which Leuckart took strong exception. *Taenia saginata* means the " stout " or " fat " tapeworm. The two respective species were first named by Rudolphi and by Goeze.

It is difficult to account for the adoption of the English term " measles ", except that the bladderworms visible in the musculature of a measly pig somewhat resemble the spotted eruption characteristic of the human disease of that name. The name is most misleading to the layman, who generally associates the parasitic disease of the pig or ox with the entirely different human disease. Not only the English, but also the French adopted a misnomer for ordinary usage in describing the disease in the pig and the ox. The French name for the disease, *Ladrerie*, is said to have been derived from the Greek *Laidros*, deformed or awkward, or from Lazareus or Lazarus, whose name has been corrupted in common speech to " Saint Ladre ". *Ladrerie* was sometimes used in former days as synonymous with " leper "and " leprous ", and it is not known by what affiliation this designation of *Ladrerie* has passed from leprosy to a parasitic affection, from which it is absolutely different. (Neumann).

How the Afrikaans word *masels*, or the immediately pre-Afrikaans South African Dutch word *mazelen* came into use is a mere conjecture. One would feel inclined to believe that measles was totally disregarded, or otherwise unobserved by our early Dutch, Huguenot and German South African ancestors. The Afrikaans word *masels* is distinctly a literal translation of the English designation of the human disease, and one is inclined, further, to theorize that measles was first observed by our English fellow-ancestors after the arrival of the 1820 settlers in South Africa, who, probably had seen measly pigs in Great Britain. In support of this theory one may cite the fact that measles in pigs was well known in Europe even in the Middle Ages, and if our first Dutch, French and German ancestors had recognized the disease in whatever pigs were slaughtered at the Cape in the early days, one feels sure that posterity would not have lost the Dutch names *finnen* and *gortigheid*, or the German *finnen* and *finnenkrankheit*, or the French *ladrerie*. Instead, in Afrikaans to-day, we use a literally translated English misnomer.

History has, therefore, unfortunately lost trace of the origin of knowledge of pig and beef measles in South Africa. Whether measles was an indigenous disease among our early native stock, or/and whether our aborigines were tapeworm carriers, is unknown. It is also unknown whether measles in pigs and cattle was first introduced into South Africa by tapeworm carriers among the early Portuguese visitors, or among subsequent Dutch, French, German and English settlers, or whether it came from the northern interior of Africa with the Bantu invasion. Nor, of course, is it known whether the infection was brought to South Africa by means of importation of infected livestock, or whether humans brought and established the " vicious circle ". Support is lent to the theory that the Bantu might have brought the disease from the north, by the fact that the Bantu invaders were closely related to the Ethiopians and Nubians, who in turn were closely associated, geographically, with the Egyptians, and the latter at least had placed a ban on the consumption of pork, for various reasons, whereas the Abyssinians were among the first to be described during the last century, as heavily infected with *Taenia saginata*.

HISTORICAL SURVEY.

That pig measles was known and recognised from the very earliest days can be learned from ancient writers. The parasitic nature of the measle, however, or its pathogenicity to man, was not known. It is hinted, however, by some authors, that the injunctions to Moses, which led to the Mosaic Laws were prompted by a knowledge that the meat of pigs, under certain conditions, might be dangerous, rather than " unclean " in the literal sense. Thus in *Leviticus* xi, 3, we read " *Whatsoever parteth the hoof and is clovenfooted, and cheweth the cud, among the beasts, that shall ye eat* "; and in the next verse: " *And the swine, though he divide the hoof and be clovenfooted yet cheweth not the cud, he is unclean to you* ". It is debated by some, as to whether Mosaic Law meant that the pig was " unclean " physically, or " unclean " due to its possible diseased condition. Vorwahl (1923), has written against this discussion, and indeed against the Bible's reference to the uncleanliness of the pig. He is of opinion that the Israelites followed the views of the Syrians, viz. the pig should be considered a sacred animal.

In Greek literature are found several references to measly pork. Thus Aristophanes, B.C. 424, in his "Comedy of the Knights", mentioned the then existing custom of examining the tongue of the pig, in order to detect the presence of the so-called "glandular tumors".

Later Aristotle (B.C. 384-323) wrote and described what we would to-day understand to be measly pigs. "Measly pigs are those which have bad meat on the shanks, neck and shoulders", he wrote, "In those parts we find most measles. When only a few measles are found the meat is sweet, but when numerous, the meat becomes watery and unpalatable. As far as we know, we only find measles disease in pigs. Sucking pigs have no measles". It was also he who first attempted a description of the symptoms: "Measles mainly appear under the tongue. Those pigs with measles appear weak in the hind quarters". Aristotle's remarkable description was almost exactly reproduced by Rufus, and later it was mentioned by Pliny, Didymaeus, Plutarch, Aretaeus, Archigenes and Androsthenes.

Von Ostertag refers to Herodotus and Plutarch, who taught us that the Egyptians were forbidden to eat pork for the reason that it produced "an excess of humors and eruptions". The Phoenicians did not eat the meat of cows or swine, but held the flesh of dogs as a delicacy. On the other hand, the Romans were extremely fond of pork. Vosgien (1911) mentions that Roman vendors of pork had to guarantee the meat against measles.

During the third century A.D., Androsthenes compared pig measles with tubercles. Aretaeus compared measly pigs with people suffering from elephantiasis.

Perhaps one of the first references to a connection between tapeworms and the ingestion of pork was suggested by Anthimus (511-534 A.D.), who wrote to Theödoric, King of the Franks, that he suspected that human beings developed tapeworms by eating raw fat pork. (Le Coultre, 1928). His suspicion was certainly not unjustifiable, although he did not know the relationship between the *cysticercus* and the tapeworm.

During the 8th Century, Pope Boniface pointed out the necessity of cooking or smoking pork thoroughly, before consumption.

The Mohammedans were also forbidden the use of pork by their Prophet, on the grounds that the pig was unclean.

Later in the Christian era we find that in Germany and in France, definite regulations regarding the inspection of pigs were framed. Von Ostertag gives very interesting historical quotations regarding meat inspection in the Middle Ages in Germany, with special reference to the treatment of measly pork. The following quotations from von Ostertag are to the point.

In the year 1261, Count Raoul IV of Neuchatel decreed that "meat showing eruptions should not be sold as good meat, and under the roof of a meat market, pork containing eruptions, or meat killed by wolves or dogs, should not be sold".

The Augsburg Charter of 1276 laid down the following most interesting regulation: " If a butcher kills a measly hog, he shall sell it to no one without a statement of this fact. All the parts of such animal shall be sold in the same booth, and if it is sold whole, it shall be only after declaration ".

In Bamberg in 1306, the City Laws forbade the sale of measly meat, and in Würzburg in 1343, punishment was enacted for " all persons who offer for sale measly and mangy meat ". In 1346 the inhabitants of the village of Wolfmannshausen agreed " to bring at an appointed time all their hogs to the Monastery of Fröwenrode, where they shall be appraised and inspected by viewing the tongues. Those, which from the appearance of the tongues, shall be considered clean and worth the estimated price, shall be retained by the Monastery ".

Similar Charters are cited for Zwickau (1348), in which the sale of measly meat was forbidden in the booths, and for Hamburg, and also for Lübeck and Stade in 1375, where it was laid down that measly meat was required to be sold in a special booth on a white cloth. In 1376 the butchers of Regensburg were punished for selling measly pigs.

In Aachen, during 1385-1386, special pig inspectors or " *Finnenkückers* " were appointed, whose instructions were to examine all pigs offered for slaughter sale, and to " brand all unclean pigs with a distinctive cut ". These pig inspectors assumed office under oath to carry out their duties fairly and scrupulously, irrespective of the social standing, race and domicile of the vendor.

In Passau in 1394, meat inspectors were appointed, whose instructions were " to throw measly pork into the Danube, and the vendor was compelled to return the price of the hog to the buyer ".

Landshüt in 1401, went a step further, and passed an Ordinance prescribing that butchers should not sell " Jew meat, or measly meat anywhere else than between the tables, and that neither Jew meat, nor measly meat should be offered as good meat ".

In Wimpfen in 1404, a Charter laid down that measly meat was to be sold in a " measly booth ", three steps removed from the ordinary meat booths. In 1414 the butchers of Ulm asked the Council to adopt the following regulations, namely regulation of the traffic in measly pork, bulls' meat and Jew meat. Whoever offered such meat for sale was not allowed to sell any other meat at the same time. " *If a butcher pickled measly pork immediately after slaughter, and the twelve sworn masters of the market were satisfied of that fact, the butcher was allowed to sell other meat* ".

Steffen von Bergendorff was made to take the oath to keep the peace in 1434, after having been imprisoned in the City of Regensburg, because he attempted to sell hogs in which the bladderworms had secretly been punctured, so that the inspector could not recognize them.

The town of Marbach in Alsace appointed sworn meat inspectors in 1437. Their duties, among others were to determine whether measly meat had been worked out into sausages.

In the year 1582, the Palatinate State Laws decreed that the meat of measly hogs, if not badly infested, should be offered for sale outside the shambles or butcher shops, at a place to be determined upon by the authorities. "In case, however, the measly meat in question is found to be quite unclean, it shall be absolutely rejected and shall not be sold, nor used."

The Slaughter Ordinances in Rostock forbade the sale of measly hogs in 1699.

The detailed directions to meat inspectors in Brüchsal about the year 1784, forbade the sale and consumption of animals suffering from certain diseases, among which *cysticercus* disease was expressly mentioned.

It will now be interesting to record that in France, in the Middle Ages, and up till the time of the French Revolution, similar Ordinances to those enacted in Germany were enforced.

In the thesis of Vosgien (1910-11), is found a number of quotations. In France, about the year 1375, specific pig inspectors were appointed. The inspectors, or *langueyeurs* had to ascertain whether pigs were measly or not, by an inspection of the pigs' tongues. A *langueyeur* could not be a butcher and an inspector at the same time. Huges Aubriot, 1375, ordained that "No one dare act as *langueyeur* until he has been proved competent by the master butchers". The duties of the *langueyeur* were fully described in the Edict of Charles VI in 1403.

The Ordinance of Robert d'Estouteville, Guard to the *Prévoté de Paris*, dated 1475, established: "No one may buy or sell, or make sausages from exhausted pigs, or measly pigs".

The *langueyeurs* were quite important personages. and held very high office. Later, they were actually appointed by the King.

According to Gach, it was decreed in France, as long ago as 1476, that measly pork could be salted for forty days, and then sold in the halls. Vosgien, however, records that in 1601 the Parliament of Paris decreed that measly pork, after having been salted for 40 days, could be sold in a specific place to be named by the *Prévot de Paris*, and had to be marked by a *drapeau blanc*, (white flag).

Vosgien also relates the punishment which was meted out to an offender. On May 28, 1716, the Chamber of Justice of Paris condemned one, Antoine Dubout, to the following punishment, because he had issued measly meat to the soldiers: "He was to be exhibited in a public place, *nu en chemise, la corde au cou,* with a burning wax candle in each hand, and with a placard on his chest and back, bearing the inscription of the nature of his offence". In addition Dubout was fined 50,000 livres, was banished from the City, and was deprived of all his rights as a butcher.

From about the middle of the 18th Century systems of meat inspection became less thorough in France and in Germany. In practising their professions correctly, qualified veterinarians had shown the public that the meat of animals suffering from certain diseases was harmless, but, unfortunately, local authorities appeared to misconstrue that teaching, and confusion resulted, to the extent that it was assumed that the meat of all diseased animals, including measly meat, was harmless. That unfortunate state of affairs continued in both Germany and France until 1852, when Küchenmeister startled helminthologists, veterinarians, medical men and hygienists, by proving that the *Cysticercus cellulosae* was the embryonic stage of the human *Taenia solium*.

HISTORICAL REVIEW OF EARLY LITERATURE AND RESEARCHES ON CYSTICERCUS-TAENIA.

Although, as has been mentioned, measles was known to the ancients, up till the year 1685 *cysticerci* were regarded as glandular tumours. During that year Hartmann discovered the parasitic nature of *cysticerci*. He described the *Cysticercus tenuicollis* as parasitic, and followed his researches further. In 1688 he recognized the animal nature of the *Cysticercus cellulosae*. Ten years later, Malpighi, having worked independently of Hartmann, confirmed the latter's work, and further described the head process of the *cysticercus* very closely. " In verminous pork, called *Lazarioli*, live numerous colonies of worms, which are the cause that the sale of such animals is forbidden by public edict ", wrote Malpighi, and continued: " These worms are in abundance in the cellular interstices of the muscle fibres of the thighs. They appear in the shape of small oblong tumours, as little sacs filled with transparent fluid, in which floats a white globular body. Should the envelope break, when pressed slightly, the worm squirts out of the vesicle, and one sees its horns coming out like those of snails. The rings fold over themselves, and the animal rolls into a ball. At the top is a little head, and on the rolled up worm there is what looks like a little umbilicus at the extremity of the vesicle ".

The animal nature of the bladderworms was not universally accepted, and confirmation of the findings of Hartmann was further effected by Fabricius and by Goeze.

In discussing the history and development of knowledge of cysticercosis-taeniasis, Leuckart repeatedly alluded to the bitter feud which, during the middle of last century, existed between himself and his equally famous co-worker Küchenmeister. To Leuckart's credit, however, he gives all honour to Küchenmeister as the scientist who established the connection between the hook-bearing *Taenia solium* and the *Cysticercus cellulosae* of the pig. Küchenmeister observed the fact that the " structure of the head and hooks corresponded so perfectly in the two forms, that the most careful investigation could establish no differences between them ". (Leuckart.)

In 1841, Steenstrup considered that the *cysticerci* could be regarded as the first step in the development of helminths, but to which they were related, he could not determine. Van Beneden followed by von Siebold, in 1850 dispelled Steenstrup's doubts. It must be pointed out, however, that prior to 1850, von Siebold maintained that *cysticerci* were tapeworms with hydropically degenerate bodies, which was due to the fact that they had developed in an abnormal host, and were not necessarily intermediate stages.

In 1854 van Beneden successfully infected a pig with bladderworms, four and a half months after he had fed the animal on *Taenia solium*. In spite of van Beneden's test, some doubt existed as to the origin of the bladderworms, and whether they actually resulted from the eggs of the tapeworm which had been administered by van Beneden. In 1855 Haubner fed single proglottides (segments) of *Taenia solium*, followed by larger pieces, at various times to three pigs. He killed the pigs, and dissected them at various periods after initial feeding, and thus established the growth of the infection with *Cysticercus cellulosae*. At about the same time, Leuckart performed a similar series of experiments on five pigs, with the same results. Later Mosler and Gerlach confirmed by further experiments the findings of van Beneden, Haubner and Leuckart.

Küchenmeister's Experiment.

This experiment caused a complete revolution in the science of meat inspection, and it is proposed to quote, in almost full detail, Neumann's description of Küchenmeister's research. Küchenmeister published the results of his experiments in 1855.

For three consecutive days prior to her execution, a condemned woman prisoner was given seventy-five *cysticerci* in her food, by Küchenmeister, who had made the necessary arrangements with the prison authorities. At the autopsy, made 48 hours after death, Küchenmeister found 10 young *taeniae*, 4 mm. to 8 mm. long, " some of which already carried several hooks ". (Neumann p. 681). Küchenmeister repeated the experiment on another prisoner condemned to death. This subject was given 20 measles on two occasions—one four months and the other two and a half months—before execution. At the autopsy he found 19 *taeniae*, eleven of which had already mature *proglottides*. Küchenmeister's experiments were repeated and the results confirmed the following year by Leuckart, who gave four fully developed *Cysticerci cellulose* in milk to a young tubercular subject, who voluntarily offered himself as a subject. Two months later, Leuckart found *proglottides* in the excretum of his subject.

With reference to historical literature on the *Cysticercus bovis* and its adult counterpart, the *Taenia saginata*, there is very little written before the 18th Century. Leuckart, however, is of opinion that the Ancient Greeks carried and encountered the *Taenia saginata* more frequently than the *Taenia solium*. He cites as evidence for his theory the fact that the former is a more common disease in Mediterranean countries and the East, than the latter disease-causing parasite; and cites too the writings of Hippocrates, " the tapeworm subject voids in portions the ripe joints of the worm "; a phenomenon, which Leuckart points out, is much more usual and striking in the case of *Taenia saginata* than of *Taenia solium*. If Leuckart's conclusions are correct, then we must believe that beef measles must, also, have been fairly common in Ancient Greek times, although they were never observed. An important factor in regard to the occurence of *Cysticercus bovis* is that the cysts are generally isolated and more frequently than not, are found singly or a mere few, in a carcass. That factor, possibly, was the reason why they were not noticed by the Ancients, or otherwise, in comparison with the usual heavy nature of the infestation of pigs, they were not considered worth while troubling about.

On similar grounds, Leuckart holds the opinion that the Arabian physicians mainly investigated *Taenia saginata.* The Arabians considered the various segments as separate entities formed into a chain, and that the chain constituted what is now known as tapeworm. These views were supported by Vallisnieri and also by Coulet at the beginning of the 18th Century, and by Linné, whose description constituted a comparison between a tapeworm and a plant of many shoots.

With regard to the older descriptions of tapeworms, we learn from Aëtius and Paulus Aeginata, that they considered the tapeworm as a metamorphic product of the intestinal mucous membrane. "*Lumbricus latus transmutatio, ut ita dicam, est membranae intestinis intrinsecus agnatae in corpus quoddam animatum.*" (Leuckart.)

Ancient Chinese Medical Views.

Gear and Pedersen (1934) give an interesting reference to the work of Chu and Chiang (1931), who translated twelve old writings, which illustrated the type of knowledge and opinion held by Chinese medicine concerning helminths. These twelve works extend over a period from Tsang Kung Lieh Chuan 180 B.C. to Pien Chiao Hsin Shu in 1767 A.D., and as Hoeppli in an introduction to the study points out, there are several remarkable similarities in the different texts. According to Gear and Pedersen, the same theories with slight variation persist through the twelve works, and are sufficiently illustrated in an extract quoted by those authors, from the translation given of the Ch'ao Shih Ping Yuan of 605 A.D.

" The ' Tsun Pai Ch'ung ' is also one of the nine worms. It is about one inch long, white in colour and flat in shape. The attack is due to the weakness of one's viscera. It is said that the infection is due to drinking of " white " wine and eating of raw beef and raw grains. It is also said that eating of raw fish followed by a drink of cold milk likewise produces the infection. It weakens one's general physical condition and produces pain and weakness of the kidney and feet. If the worm grows to one foot in length it causes the death of the host."

Gear and Pedersen quote Hoeppli, who says: " It is very remarkable, however, that several times the ' Ts'un Pai Ch'ung ' which, in our opinion, is a tapeworm, is said to be produced by eating raw meat, a belief which is evidently not purely speculative. but rather the result of observation ".

Actual beef measles, had, however, been seen for many years, but they were considered as tumours. In 1684, Redi in Italy and Hartmann and Wepfer in Germany recognized the animal nature of the larvae from their movements and organisation. (von Ostertag). In 1767 Linnaues and in 1781 Pallas saw parts of *Taenia saginata*, but in 1782 Goeze described the parasite.

Perhaps Knox, who served in South Africa as an Army Surgeon, might be considered as one of the first writers who connected an outbreak of tapeworms among humans with the ingestion of beef. Edmonds (1922) refers to Knox's report of an outbreak of tapeworms among soldiers who had participated in the Kaffir War in 1819. Knox ascribed the cause of the infection as due to the fact that " the soldiers had eaten the meat of oxen which had been driven too fast, and were exhausted ". Leuckart states that he examined some specimens of the Cape tapeworm, which were sent to him, and he was satisfied that they were *Taenia saginata.* Leuckart makes special reference to " Knox's outbreak ", and strongly hints that this outbreak and its association with the ingestion of beef and certain other factors were instrumental in causing the experiments he conducted in 1861, which definitely established the relationship between *Taenia saginata* and *Cysticercus bovis*. The various factors which Leuckart considered were the reports of ancient and modern travellers, and particularly of Duvaine in 1860, that from earliest times, almost without exception, the Abyssinians who ate no pork were heavily infested with tapeworms; that Jews and Mohammedans, who likewise ate no pork, were frequently infested with tapeworms; and lastly, Weisse's report from St. Petersburg in 1857, that he had often fed raw beef to delicate children, and that tapeworms had frequently been developed in his patients. Leuckart, also, very fairly, mentioned that Hüber and Schmidt had already noted the probability that the ox could be the intermediate host of *Taenia saginata.* The latter had mentioned to Leuckart that with some certainty he had traced the existence of *Taenia saginata* to the ingestion of a meat salad made from raw beef.

Leuckart's Experiment.

Describing his experiment, Leuckart states that in November 1861, he gave about a yard of some 80 ripe segments of *Taenia saginata* to a calf four weeks old, and about 8 days later he repeated the feeding with a smaller dose. He mentions that the animal he experimented on seemed to be so slightly affected by the experiment, that he was about to extract a muscle, when, 25 and 17 days after the first and second feedings, respectively; he found the calf dead. On post-mortem examination he found all the muscles, and especially those of the breast and neck, and the psoas, had been penetrated by cysts, which measured about 2 to 4 mm. by 1·5 to 3 mm. in size. He found those cysts numerous everywhere, " and in many places they lay so thickly together, that their total number must have been many thousands, yet, it seemed at first as if the death of the animal under experiment could hardly have been caused by them ", writes Leuckart. " It was, however, indeed the *cysticerci* which had killed the calf. Further examination showed that the distribution of the parasites was in no way confined to the peripheral muscles of the body ", he concludes.

Among other localities Leuckart found " crowds of cysts followed the course of the swollen lymphatic vessels and glands into the inguinal region." " Some of the glands were not only reddened, but were full of extravasated blood, which permeated throughout their entire mass ", continues Leuckart; and he concludes, " I had almost no scruple in referring the death of the animal to the pathological state of inflammation of the lymphatics. The latter may also be traced to the state of inflammation which resulted from the immigration and development of such number of parasites."

Leuckart's colleagues Seitz and Mosler agreed with his views and " so have all my successors concluded ", writes Leuckart, " except Küchenmeister ". Leuckart bitterly quotes the writing of Küchenmeister: " Leuckart's first experiment, taken by itself teaches us nothing, except that, after abundant feeding with the *proglottides* of *Taenia mediocannelata* (Küchenmeister's nomen for *T.saginata*), the animal remained long, apparently unhurt, till suddenly, 25 days after feeding it died and exhibited a miliary tuberculosis caused by the Cestode brood. Without the subsequent experiments, I cannot regard the first as of special value in regard to *Taenia mediocannelata* ".

On December 27th, that year, Leuckart repeated the experiment on a second calf, but remembering the severe results of his first experiment, and the resultant death of the calf, he used smaller doses of segments, and repeated these at five to six day intervals, until the calf received about 50 segments. Twenty days after the first infection, many pathological phenomena appeared, for example, loss of appetite, fatigue, ruffling of the hair and fever, but those clinical signs subsided, till finally perfect health returned. Forty-eight days after the first, and thirty days after the last feeding, Leuckart extracted the sterno-hyoid muscle of the left side. In this muscle he found about a dozen cysts. The cysts were of various size, representing various stages of development. In examining those embryos, Leuckart made the striking discovery that although the bladderworms were the " descendants and young forms of a hookless tapeworm, they were furnished with a distinct, though small *rostellum*, and with the rudiments of hooks ". Later, Leuckart watched the development of the *cysticerci* by extracting other muscles at various periods. He thus proved, conclusively, that one of man's principal food animals, the ox, was the intermediate host of the human tapeworm *Taenia saginata*. All but one pair of the following list of subsequent investigators obtained positive results from confirmatory experiments:—

Germany.— Mosler (1864); Röll (1865); Gerlach (1869); Zürn (1871); Zenker (1872); Probstmayr (1879).

France.— St. Cyr (1873); Masse and Pourquier (1877).

Belgium.— van Beneden Junior (1879).

Italy.— Perroncito (1877).

England.— Simonds and Cobbold (1866)—negative results.

Commenting on the report of Gerlach, Leuckart states, *inter alia* " Gerlach killed his experimental calf 5 months after feeding, and found that it was penetrated through and through with bladderworms ".

Conversely, shortly afterwards, Oliver and Perroncito infected themselves and their respective assistants with *Taenia saginata*, after having ingested *Cysticercus bovis* bladderworms.

PART II.

A Survey of the Incidence of Cysticercosis in Swine and Bovines.

In providing a survey of this nature, it must be explained that figures representative of the incidence of infection as observed at abattoirs in many countries, must be regarded as not necessarily indicative of the actual extent of infestation in such particular countries, since, in many cases infected stock slaughtered may have been imported from elsewhere, and the survey would then, rather, tend to show the surmised incidence of infection in the export country. In some countries also, reliable statistics have not been compiled, and, therefore, data given must frequently be judged more as speculative than actual. In some other countries statistics of infection were available many years ago, but more recently, owing to a decreased incidence of infection, the relatively few cases have not been recorded.

The statistics supplied in the following pages have been obtained from (*a*) old, recent and contemporary literature; (*b*) as the result of personal enquiry from the respective authorities and from the obliging replies sent by those colleagues; (*c*) from " speculative " sources reflecting the incidence of infection found among exported stock slaughtered and found infected in foreign countries. It might be explained that questionnaires were forwarded to no less than fifty countries. and replies were obtained from the vast majority of them. It is regretted that no statistics are available for a few important territories.

A. THE INCIDENCE OF CYSTICERCOSIS IN SWINE AND BOVINES IN EUROPE.

Great Britain.

Through the kind favour of Col. T. Dunlop Young of London, enquiries were made from the abattoir reports from most of the important centres, but not a single instance of measles was reported for the year 1935.

Robertson (1920) accidentally infected some if his patients at Leith (whom he had placed on a raw beef diet for tuberculosis) with *Taenia saginata*. It can, therefore, be presumed that *C. bovis* must have occurred at that time among Scottish slaughter cattle.

Stockman (1909) stated that although no statistics on the frequency of measles were available in Britain, there was little doubt that it existed in British swine at that time. " In the past few years the author has met with several cases, and others have been reported by practitioners." (Stockman, 1909). Cameron (1933) suggests that

Germany.

According to von Ostertag (1934), the incidence of *C. bovis* in oxen varied between 0·321 per cent. in 1904 and 0·27 per cent. in 1928. In Berlin the percentage of animals infected fell from 0·84 in 1913 to 0·33 in 1922, and rose to 0·617 in 1928. In Breslau, Mahlendorff (1930) recorded the incidence of infection at that abattoir during 1929-30 to be over 1 per cent. The highest percentage (1·54) was during the month of November, 1929 (i.e. during the period under report).

According to Leeb and Berngrüber (1932), during 1931, 1·906 per cent. of slaughtered bovines were found to be measly in Würzburg, and there was evidence that infection was increasing throughout the State of Bavaria. According to Krueger (1934), 2 per cent. of all cattle slaughtered in Kottbus were infected with *C. bovis*.

Junack (1926) draws attention to the fact that for Prussia for the years 1922 and 1923, 18 and 11 bovines, respectively, were shown as measly, whereas the thousands of cattle which were passed after treatment (cooling for 21 days), were not mentioned. Thus the last-named numbered 403 and 398 for Berlin alone. Junack mentions that, by not including all these lightly infested bovines in the count, a false impression is given. Thus, on the one hand hygienists, and on the other hand butchers get the wrong impression that *C. bovis (inermis)* is not of much moment any longer from a point of view of Public Health and Food Economy.

In Germany the incidence of *C. cellulosae* has diminished to almost nil in German-reared pigs. Most of the cases found during the past few years at German abattoirs have been in imported slaughter pigs. Thus Berdel (1930) records that at Frankfurt a.M. abattoir, between 10.9.29 and 19.11.29, out of 1,415 pigs imported from Lithuania, 100 were found to be measly (i.e., 47 heavily infested and 53 lightly infested). The same author quotes Meyer, who stated the year before that 19·47 per cent. of slaughtered Russian pigs were found to be measly at Barnaul. In the Saxon foreign-import meat inspection halls 39 out of 13,472 half-pigs were found measly during 1925-26. (Berdel.)

Von Ostertag (1913) showed the gradual diminution in the number of measly hogs in Germany thus:—

(a) *Kingdom of Prussia.*		(b) *Kingdom of Saxony.*		(c) *Berlin.*	
	Percentage.		*Percentage.*		*Percentage.*
1876–1882	0·324	1894	0·151	1883–1890	0·577
1886–1889	0·181	1896	0·017	1892–1893	0·319
1890–1892	0·122	1899	0·010	1895–1896	0·099
1899	0·09			1899	0·043

Ministerialdiregent Professor Dr. Müssemeier of Berlin kindly supplied the following official table showing the incidence of cysticercosis (measles) in Germany for the ten years, 1925-1934:—

Year.	Heavily infested.				Lightly infested.			
	Cattle.		Pigs.		Cattle.		Pigs.	
	No.	Per/1000.	No.	Per/1000.	No.	Per/1000.	No.	Per/1000.
1925......	112	0·03	193	0·02	6,174	1·91	336	0·03
1926......	102	0·03	178	0·01	6,801	2·09	256	0·02
1927......	103	0·034	92	0·01	7,110	2·23	186	0·01
1928......	166	0·05	98	0·01	9,555	2·69	222	0·01
1929......	167	0·04	567	0·03	11,257	2·82	646	0·04
1930......	194	0·05	753	0·04	11,501	3·24	975	0·05
1931......	278	0·06	219	0·01	11,950	3·53	283	0·01
1932......	247	0·06	60	0·00	135,368	3·83	133	0·01
1933......	224	0·06	49	0·00	141,188	4·08	131	0·01
1934......	350	—	46	—	16,697	—	108	—

Note the steady increase in numbers and per thousand in cases of *C. bovis* in Germany.

Switzerland.

The statistics given for Switzerland were kindly obtained by Dr. W. Frei, of the Veterinary Pathological Institute of the University of Zürich, from the abattoirs at Basel, Zürich and Berne. The statistics cover periods ranging from 15 years to 25 years, and may be taken as fairly representative for Switzerland. The statistics are those for pigs and adult bovines only. The incidence of *C. bovis* in calves is very low.

At the Abattoir at *Berne*. Statistics kindly supplied by the Director:—

Year.	Bovines slaughtered.	Measly.	Percentage.	Year.	Bovines slaughtered.	Measly.	Percentage.
1921.....	4,175	16	0·38	1930....	5,487	18	0·30
1932.....	5,116	16	0·31	1931....	4,788	20	0·42
1924.....	6,205	18	0·29	1932....	4,745	34	0·72
1925.....	5,098	7	0·14	1933....	5,650	21	0·37
1926.....	4,615	8	0·17	1934,....	7,202	17	0·23
1927.....	4,799	18	0·38	1935....	8,518	15	0·18
1928.....	5,012	9	0·18	1936....	5,496	10	0·18
1929.....	4,974	13	0·26				

Very nearly half the cattle killed at Berne were imported.

NOTE.—During the above period only one measly pig was slaughtered (during 1926, out of over 20,000). This pig was imported from Italy.

At the Abattoir at *Zürich*. Statistics kindly supplied by the Director:—

Year.	Bovines killed.	Measly.	Percentage.	Year.	Bovines killed.	Measly.	Percentage.
1910.....	11,838	20	0·17	1923....	12,929	14	0·11
1911.....	11,181	24	0·21	1924....	17,629	6	0·034
1912.....	10,918	20	0·18	1925....	14,902	17	0·12
1913.....	11,150	15	0·13	1926....	12,660	21	0·17
1914.....	11,835	26	0·22	1927....	20,042	14	0·069
1915.....	5,177	8	0·15	1928....	12,011	13	0·11
1916.....	13,369	24	0·18	1929....	19,740	21	0·106
1917.....	13,558	32	0·24	1930....	13,477	9	0·067
1918.....	20,015	44	0·22	1931....	7,920	3	0·038
1919.....	18,062	27	0·15	1932....	9,854	4	0·041
1920.....	10,994	13	0·12	1933....	17,036	15	0·088
1921.....	3,946	6	0·15	1934....	17,569	9	0·051
1922.....	11,531	19	0·16	1935....	11,340	10	0·088
				1936....	15,575	16	0·13

During the period 1910-36, over half-a-million pigs were slaughtered, of which number only 52 were measly.

At the Abattoir at *Basel*. Statistics obtained from annual reports for the years 1915 till 1935. Reports kindly supplied by Dr. J. Unger, Director of Abattoirs:—

Year.	Bovines.	Measly.	Percentage.	Year.	Bovines.	Measly.	Percentage.
1913.....	18,285	15	0·082	1925....	13,663	72	0·52
1914.....	16,639	15	0·09	1926....	13,770	22	0·16
1915.....	14,546	24	0·17	1927....	13,045	40	0·31
1916.....	12,621	17	0·14	1928....	13,618	32	0·23
1917.....	13,402	12	0·09	1929....	14,732	39	0·27
1918.....	17,455	45	0·26	1930....	12,720	39	0·31
1919.....	14,211	61	0·43	1931....	13,388	49	0·37
1920.....	10,221	61	0·61	1932....	13,975	40	0·29
1921.....	9,807	27	0·28	1938....	15,425	13	0·29
1922.....	11,858	39	0·33	1934....	16,485	38	0·23
1923.....	14,224	52	0·37	1935....	16,533	24	0·15
1924.....	18,167	100	0·55				

During the period quoted above more than one million pigs were killed at Basel, and of that number only eleven were found measly, the last (one pig) being in 1931, and previous to that, one pig in 1924.

Special Notes relative to the Reports for the Abattoir for Basel:—

1920: Of 61 measly cattle, 11 were imported, viz., from *Denmark* 6 out of 1,165; from *Canada* 2 out of 615; from *Italy* 2 out of 222; from *Lichtenstein* 1 out of 52.

1921: of 27 measly cattle, 16 were imported, viz., from *Denmark* 7 out of 2,427; from *Czechoslovakia* 5 out of 1,228; from *Canada* 4 out of 1,145.

1922: Of 39 measly cattle, 13 were imported, viz., from *Denmark* 1 out of 439; from *Germany* 1 out of 93; from *Canada* 1 out of 152; from *France* 3 out of 43; from *Czechoslovakia* 5 out of 788; from *Argentine* 2 out of 770.

1923: Of 52 measly cattle, 28 were imported, viz., from *Holland* 1 out of 191; from *South West Africa* 1 out of 204; from *Argentine* 8 out of 908; from *Denmark* 18 out of 5,024.

1924: Of 100 measly cattle, 87 were imported, viz., from *Canada* 1 out of 392; from *South West Africa* 2 out of 117; from *Czechoslovakia* 17 out of 235; from *Germany* 8 out of 1,340; from *Argentine* 11 out of 3,742; from *Denmark* 58 out of 8,833.

1925: Of 72 measly cattle, 56 were imported. viz., from *Austria* 19 out of 1,502; from *Italy* 14 out of 1,394; from *Canada* 8 out of 2,321; from *Czechoslovakia* 7 out of 624; from *Germany* 5 out of 539; from *Hungary* 3 out of 1,821.

1926: Report does not give separate origin of measly stock.

1927: Of 40 measly cattle, 19 were imported, viz., 14 out of 411 from *Czechoslovakia;* 2 out of 2,713 from *Hungary;* 3 out of 991 from *France.*

1928: Of 32 measly cattle, 4 were imported, namely from *France* 2 out of 1,910; from *Hungary* 2 out of 480.

1929: No stock imported.

1930: Of 39 measly cattle, 16 were imported, all from *Hungary*, i.e., 16 out of 2,889.

1931: Of 49 measly cattle, 23 were imported, viz., from *Germany* 1 out of 233; from *Hungary* 22 out of 3,814.

1932: Of 40 measly cattle, 3 were imported, viz., 1 out of 376 from *Hungary*; 2 out of 56 from *Czechoslovakia.*

1933-1935: No records of imported cattle.

In older Swiss literature, Buri (1915) mentioned that in Eastern and North-Eastern Switzerland the incidence of *C. bovis* was higher than in Western Switzerland. Thus, for Eastern Switzerland he gave an incidence of 1·5 to 2·3 per cent., and for Western Switzerland 0·3 to 0·4 per cent.

Krupski (1917) found at Liestal a percentage of 5·9. This high percentage Krupski attributed to more thorough inspection of predilection sites.

Holland.

Le Coultre (1928) obtained the following data from Professor van Oijen:—

At *Rotterdam* from 1918 to 1923, only cases with living *Cysticerci bovis* were noted. The percentage infection varied between 0·001 and 0·003. From 1924 to 1927 cases with degenerated measles were also noted, and the percentage was then between 0·1 and 0·2.

At *Haarlem*, in ten years up to 1927, the incidence varied between 0·33 per cent. and 0·6 per cent.

At *Alkmaar* the incidence varied between 0·1 per cent. and 0·5 per cent.

At *Leiden*, in adult bovines, between 1918 and 1922 the incidence of infection varied between 0·1 per cent. and 0·66 per cent.; in 1923 it was 0·04 per cent.; and between 1924 and 1927 it varied between 0·2 per cent. and 0·4 per cent.

At *Groningen*, in ten years the incidence varied between 1·03 per cent. and 1·5 per cent.

At *Arnhem*, the figures were: 1918, 1·51 per cent.; 1919, 2·45 per cent.; 1920, 2·94 per cent.; 1921, 3 per cent.; 1922, nearly 4 per cent. (238 cases out of 5,927 bovines slaughtered); from 1923 to 1927 the percentage varied between 2 and 2·75 per cent.

At *Nijmegen*, the percentage varied from 1918 tot 1922, between 0·22 and 0·4. From 1923 to 1927 a sudden tremendous increase in the percentages occurred, thus for the five years 1923-1927, inclusive, the figures were 3 per cent.; 3·7 per cent.; 4·7 per cent.; 4·4 per cent. and 3·2 per cent., respectively.

[Le Coultre ascribes this increase in the incidence, as observed at Arnhem and Nijmegen to more thorough inspection technique. School (1933) expressed a similar opinion.]

At *Utrecht* the percentages varied from 1918 to 1927 between 0·23 and 0·61.

Recent Statistics.

Professor C. F. van Oijen of Utrecht kindly supplied me with the following statistics for the years 1933 and 1934:—

Cysticercus bovis was found in 1933 in 4,515 adult bovines, and in 1934 in 4,572 adult bovines. According to Prof. van Oijen, the number of cases of cysticercosis, so far as this concerns the whole country, has again risen, namely from 0·83 per cent. to 0·92 per cent.

At *Leeuwaarden* an increase in the number of cases of cysticercosis in bovines has been noted, which is reflected in the following statistics:—

	Dead Specimens percentage cases.	*Living Specimens percentage cases.*
1930	0·69	0·016
1931	1·07	0·059
1932	1·74	0·06
1933	1·6	0·06
1934	1·64	0·1

At *Rheeden*, 9·2 per cent. of the total slaughtered bovines were found measly—105 cases. "The percentage is still steadily increasing". (Prof. van Oijen.)

At *Arnhem*, *Cysticercus bovis* was found in 257 adult bovines (4·45 per cent.). "Percentage is still increasing". (Prof. van Oijen.)

At *Utrecht*, 128 adult bovines were found measly. The percentage this figure represents was not given.

At *Apeldoorn*, 64 adult bovines were found measly (2·1 per cent.).

At *Zutphen*, 138 adult bovines were found measly (4·24 per cent.).

At *Doetinchem*, in adult bovines 4·07 per cent. were found measly. (The number of cases has increased.)

At *Amersfoort*, 17 cases were measly (percentage not given).

At *Amsterdam*:

1st quarter, 9 cases living or 0·08 per cent. and 24 cases dead measles, 0·32 per cent.

2nd quarter, 7 cases living or 0·07 per cent. and 18 cases dead measles, 0·18 per cent.

3rd quarter, 8 cases living or 0·09 per cent. and 47 cases dead measles, 0·52 per cent.

4th quarter, 20 cases living or 0·17 per cent. and 93 cases dead measles, 0·75 per cent.

At *Haarlem*, where the percentage cysticercosis is considerably higher than at Amsterdam, the increase was not so obvious, as is shown in the subjoined table:—

1st quarter, 10 cases living measles—0·45 per cent. and 37 cases dead measles—1·7 per cent.

2nd quarter, 7 cases living measles—0·3 per cent. and 38 cases dead measles—1·6 per cent.

3rd quarter, 8 cases living measles—0·33 per cent. and 55 cases dead measles—2·3 per cent.

4th quarter, 7 cases living measles—0·28 per cent. and 62 cases dead measles—2·5 per cent.

Only three cases of *C. cellulosae* were found in pigs in Holland during the years 1933 and 1934. Kerstens (1931) showed that it was dangerous to presume that *C. cellulosae* was non-existent in Holland. He referred to a case he found in a pig which was slaughtered domestically by a farmer.

Belgium.

Professor V. Rubray, Rector of the *Ecole de Mèdicine Vètèrinaire*, Cureghem-lez-Bruxelles, writes (5th March, 1937):-

" 1. As regards infection of the pig, we find only one or two cases per year, out of about 150,000 subjects slaughtered at the abattoirs.

2. In cattle, during the war, 1 to 2 per cent. were found infected, but nowadays it is as rare as in the pig.

The result of this notable decrease in the incidence of cysticercosis we attribute to our hygienic measures and the fact that the ox and the pig are given no facility to become contaminated by human excretum."

France.

In spite of exhaustive enquiry into recent French literature, and personal communications to French authorities, the present author was unable to obtain any recent information as to the incidence of *C. cellulosae* and *C. bovis* in France at the present time.

Vosgien (1911) gave the percentages recorded at three centres. *C. cellulosae* in pigs:—

Paris: 1900, 0·03 per cent.; 1901, 0·05 per cent.; 1904, 0·01 per cent.; 1906, 0·0175 per cent.; 1910, 0·034 per cent.

Bordeaux: 1905, 0·05 per cent.; 1906, 0·063 per cent.; 1909, 0·024 per cent.

Limoges: 1890, 0·76 per cent.; 1895, 0·62 per cent.; 1900, 0·48 per cent.; 1905, 0·41 per cent.; 1910, 0·27 per cent.

According to Ballon (1913) (le Coultre, 1928), the percentage infected bovines was as high as 17·42 (i.e. 23 cases out of 132 bovines) at Troyes-sur-Aube. Raymond (le Coultre, 1928) found the percentage to be 3·5 in bovines in Paris in 1908 and 1909.

If we were to consider the incidence of infection in French bovines exported to Switzerland as a criterion of the extent of infection in French domestic cattle, then the incidence of *C. bovis* in that country is very much lower at the present time. During the years 1922-32, 3,140 cattle from France were slaughtered at the abattoir at Basel, Switzerland, and of this number only 8 were infected (0·25 per cent.). The maximum record of infection was shown in 1922, when 3 French cattle out of 43 were found measly at Basel. (Approximately 7 per cent.)

Spain.

Owing to conditions at the present time, it is not possible to obtain statistics from this country. According to Vosgien (1910-11), 0·29 per cent. of pigs slaughtered in Madrid in 1910 were found to be measly. Out of 61,457 pigs slaughtered in that city during 1910, 180 had measles.

Portugal.

Dr. Fernando de Fontes Pererira de Mello kindly supplied the following statistics relevant to the incidence of cysticercosis in Portugal:—

1. *Cysticercus cellulosae*—(Portugal).

	1933	1934	1935
Number of cases	312	429	437
Percentages	0·184%	0·209%	0·213%

2. *Cysticercus bovis*—(Lisbon abattoir).
Number of cases: 153.
Percentages of slaughtered bovines: 0·003 per cent.

These cases came from:—

Alentejo (Portugal)	1
Ribatejo (Portugal)	1
Angola (West Africa)	151

Italy.

Cysticercus cellulosae was relatively common in Italy about the year 1870. Thus, Pellizari (Leuckart, 1886) estimated the number of measly pigs in Italy to be 1 per 3,000, but Perroncito (Leuckart) stated that in Turin 1 pig in every 250 was measly, and in Milan 1 in every 70.

In reply to a request for information on the present incidence of cysticercosis in Italy, the Union Minister Plenipotentiary at Rome very kindly submitted the following translated *Note Verbale* dated 17th March, 1937, from the Royal Italian Ministry of Foreign Affairs.

Note Verbale (17.3.37).

> " In Italy, due to the continuous and strict reinforcement of the Legislative Regulations dealing with sanitary supervision over meat, infection through *Cysticercus cellulosae* has become rare, so much so that in many big abattoirs in the Kingdom, where the meat of thousands of pigs has been controlled for many years, there has not been found a single case.
>
> The same thing can be said about *Cysticercus bovis*, which, for example, has for more than 10 years not been found in the Rome abattoir, notwithstanding the continuous and regular research as with *Cysticercus cellulosae*, in points where the infection is most likely to be found.
>
> It should also be considered that in Italy butchering for private use is, by regulation, under veterinary control, and it is to be borne in mind that this too, has advantageously contributed to reaching the favourable situation indicated above ".

From 1920 to 1932, 16 export Italian bovines out of 1,837 were found measly at the abattoir at Basel (Switzerland).

Austria.

Vosgien (1910-11) gives the following statistics for the Vienna abattoir:—

In 1902: 4,109 cases of measles out of 594,539 pigs slaughtered —0·671 per cent.

In 1903: 3,425 cases of measles out of 564,813 pigs slaughtered —0·606 per cent.

In 1904: 3,213 cases of measles out of 579,317 pigs slaughtered —0·555 per cent.

In 1905: 4,243 cases of measles out of 575,340 pigs slaughtered —0·737 per cent.

In 1906: 3,421 cases of measles out of 600,244 pigs slaughtered —0·569 per cent.

In Wiener-Neustadt, Schmidt (von Ostertag, 1913) found between the years 1901 and 1910 that 1·8 per cent. of pigs were measly.

According to Schmid (1930), 89 pigs out of 54,461 slaughtered at Wiener-Neustadt (i.e., 0·17 per cent.) were found measly in 1929. Of this number 57 came from Yugoslavia; 16 from Hungary; 11 from Poland.

According to the same author, 17 bovines out of 5,439 were measly at Wiener-Neustadt during 1929.

The *Chef der Veterinärverwaltung des Oesterreichischen Bundesministerium für Land-u.Forstwirtschaft* writes (letter dated 8.1.37): "In the years 1930 to 1935 were found in the abattoir of the Capital City of Vienna:—

1930: *Cysticercus cellulosae* in 2,983 pigs out of 696,233 slaughtered—0·43 per cent.

1931: *Cysticercus cellulosae* in 2,441 pigs out of 860,707 slaughtered—0·28 per cent.

1932: *Cysticercus cellulosae* in 2,702 pigs out of 711,932 slaughtered—0·38 per cent.

1933: *Cysticercus cellulosae* in 2,153 pigs out of 687,660 slaughtered—0·31 per cent.

1934: *Cysticercus cellulosae* in 967 pigs out of 735,244 slaughtered—0·13 per cent.

1935: *Cysticercus cellulosae* in 793 pigs out of 647,678 slaughtered—0·12 per cent.

1930: *Cysticercus inermis* (*bovis*) in 73 cattle out of 129,050 slaughtered—0·057 per cent.

1931: *Cysticercus inermis* (*bovis*) in 120 cattle out of 128,463 slaughtered—0·094 per cent.

1932: *Cysticercus inermis* (*bovis*) in 155 cattle out of 130,449 slaughtered—0·12 per cent.

1933: *Cysticercus inermis* (*bovis*) in 114 cattle out of 108,895 slaughtered—0·104 per cent.

1934: *Cysticercus inermis* (*bovis*) in 156 cattle out of 105,852 slaughtered—0·14 per cent.

1935: *Cysticercus inermis* (*bovis*) in 217 cattle out of 113,874 slaughtered—0·19 per cent.

During the years 1901 to 1928, in Wiener-Neustadt, 8,697 pigs out of 1,101,544 slaughtered, were found to be measly, i.e. 0·79 per cent. Between the years 1926 and 1928, in Wiener-Neustadt, among slaughtered bovines 0·14 per cent. of the cattle from Lower Austria, 0·49 per cent. from the Burgenlands, 0·189 per cent. from Hungary and 0·49 per cent. from Roumania were found to be measly".

The *Chef* then stresses the point that one should observe that in Vienna a large number of the slaughtered pigs and cattle comes from the neighbouring states, Yugoslavia, Hungary, Roumania and also from Poland. This also applies to slaughter pigs in Wiener-Neustadt.

According to the various annual reports for the abattoir at Basel, Switzerland, Dr. Unger found, between 1920 and 1932, that 20 out of 1,595 bovines imported from Austria (including Lichtenstein) were measly.

Hungary.

According to Brener (Vosgien), the statistics of *C. cellulosae* at Budapest abattoir during the years 1902-1905 showed that 10,265 pigs out of 987,908 slaughtered, were measly, that is 1·03 per cent.

These pigs were analysed as follows, as regards origin:—

0·64 per cent. of Hungarian pigs were measly.
3·91 per cent. of Croatian pigs were measly.
2·26 per cent. of Serbian pigs were measly.

Judging from his observations at Wiener-Neustadt, Schmid (1930) estimated that between 1926 and 1928, 0·189 per cent. of Hungarian cattle were measly. This percentage represented the Hungarian export cattle which were found measly at Wiener-Neustadt.

During the years 1920-1932, 12,093 Hungarian cattle were slaughtered at Basel, Switzerland. Of this number 46 were found to be measly, approximately 0·38 per cent. The highest number was 22 out of 3,814 in 1931. (*Jahresbericht des Schlachthofes von Basel-pro* 1920 *bis* 1932.)

Czechoslovakia.

In 1896 Prettner found that 3·44 per cent. of pigs slaughtered at Prague were measly. In 1902 it was found that 1,823 cases were measly out of 356,579 pigs slaughtered at Prague, that is 0·51 per cent.

In 1909 in Dux, Liebscher found *C. cellulosae* in 2 per cent of pigs, and in 1910 in 1 per cent. of pigs. In both years *C. bovis* was found in 0·6 per cent. of cattle by Liebscher.

For Karlsbad, Messner (1930) shows that the incidence of *C. bovis* during the twenty-five years, 1905-1929, had fallen from 2·6 per cent. and 3·0 per cent. in 1905 and 1906, respectively, to 0·44 per cent. in 1916. The following year it increased to 1·4 per cent., but fell suddenly to 0·2 per cent. in 1918. Then, between the years 1919 and 1922, the percentage oscillated round about 1·1. Between 1923 and 1927 the percentage varied from just below 0·5 to 0·8. In 1928 it rose to 1·44 per cent. and in 1929 it was 2·55 per cent., the third highest record during the 25 years under report.

During the years 1920-1932, forty bovines from Czechoslovakia, out of 3,961 slaughtered at Basel, Switzerland, were measly, that is over 1 per cent. Of this number, 14 out of 411 were found to be measly in 1927. Dr. Unger, Director of the Basel Abattoir, made special mention of this record percentage (3·4) in consignments from a single country. (*Jahresbericht des Slachthofes von Basel-Stadt pro* 1927.)

In a letter dated 3rd February, 1937, the Czechoslovak Republic Ministry of Agriculture supplies the following statistics in respect of the incidence of cysticercosis at their three principal abattoirs:—

Abattoir.	Year.	Slaughtered.		Cysticercosis found.			
		Bovines.	Swine.	Bovines.		Swine.	
		No.	No.	No.	%	No.	%
Prague..........	1930	—	434,427	—	—	802	0·18
	1931	—	366,471	—	—	306	0·083
	1932	—	374,711	—	—	742	0·19
	1933	—	263,615	—	—	226	0·036
	1934	—	333,915	—	—	93	0·028
	1935	67,796	381,090	70	0·103	223	0·103
	1936	57,629	370,638	174	0·302	333	0·089
Brno............	1930	18,804	50,321	2	0·011	12	0·024
	1931	16,864	55,368	—	—	1	0·0018
	1932	18,471	51,706	1	0·0054	7	0·013
	1933	16,253	41,100	2	0·012	17	0·041
	1934	18,062	43,345	5	0·028	3	0·006
	1936	17,911	56,991	4	0·022	1	0·001
	1936	14,711	55,160	1	0·0067	8	0·014
Bratislava.......	1930	9,915	50,645	2	0·0201	1	0·002
	1931	9,999	57,430	1	0·01	4	0·007
	1932	10,727	55,924	3	0·027	3	0·0052
	1933	10,304	48,379	2	0·019	8	0·017
	1934	10,927	55,324	10	0·092	3	0·006
	1936	10,150	56,914	8	0·076	—	—
	1936	9,250	56,669	36	0·39	3	0·005

Yugoslavia.

It is not clear what the extent of infection with *C. cellulosae* and *C. bovis* is at the present time in this multi-raced Kingdom.

Between the years 1902-1905, Brener found that 3·91 per cent. of the Croatian pigs and 2·26 per cent. of the Serbian pigs were found measly in the abattoir at Budapest (Hungary).

According to Vosgien, cysticercosis is quite rare in Croatia and Slavonia, but very common in Serbia. Martel, according to Vosgien, found in 1905 that from 8 per cent. to 12 per cent. of Serbian pigs were measly. In Bukowina and in Dalmatia, figures of 6 per cent. and 5 per cent., respectively, are given.

Schmidt, according to von Ostertag, found 0·83 per cent. of Croatian pigs to be measly at Wiener-Neustadt, Austria, between 1901 and 1910.

According to Kukuljevic (1906) 0·5 per cent. of pigs in Serbia were found to be measly in tongue-inspections, without resorting to meat inspection. Kukuljevic attributed the high incidence of measles in pigs in Serbia to the unhygienic customs in that country, where pigs are allowed to wander about the streets and on open fields, and thus greater facility for infection existed than would have been the case had proper stying and husbandry been practised.

Rumania.

Schmidt (1930) found that between 1926 and 1928 0·49 per cent. of Rumanian cattle slaughtered at Wiener-Neustadt, Austria, were measly.

The Director of the *Directiunea Zootechnica si Sanitara Veterinaria* kindly supplied the following official statistics showing the recentmost incidence of *C. cellulosae* and *C. bovis* as observed in Rumania:—

Year.

1933: 5,981 cases, i.e., 1·05 per cent. of the total pigs slaughtered at the abattoirs were measly.
110 cases, i.e. 0·014 per cent. of the total bovines slaughtered at the abattoirs were measly.

1934: 7,984 cases, i.e., 1·25 per cent. of the total pigs slaughtered at the abattoirs were measly.
139 cases, i.e., 0·018 per cent. of the total bovines slaughtered at the abattoirs were measly.

1935: 4,604 cases, i.e., 0·77 per cent. of the total pigs slaughtered at the abattoirs were measly.
168 cases, i.e., 0·018 per cent. of the total bovines slaughtered at the abattoirs were measly.

Bulgaria.

Dikoff (1931) mentioned in his article that as regards eradication of taeniasis, Bulgaria had yet to commence, and a good deal had yet to be accomplished in meat inspection. According to Dikoff, the actual extent of human infection with *Taenia solium* and *Taenia saginata* is not known in that country, but *C. cellulosae* is encountered on an average in 0·39 per cent. to 2·45 per cent. in Bulgarian pigs. Since 1920 Dikoff has noticed no decrease in the incidence. Pig dealers know the disease, and frequently bring pigs to slaughter houses where no inspections exist, rather than risk condemnation at properly controlled abattoirs.

Cysticercus bovis, according to Dikoff, is very common. In Schumen the incidence of infection is 2·97 per cent. in adult bovines (buffaloes) and 5·8 per cent. in calves.

Russia.

According to von Ostertag, *C. cellulosae* is a common disease in Russian pigs. Thus, Menzel, according to von Ostertag, mentioned that during 1904 and 1907, 1·68 to 3·21 per cent. of Russian pigs imported into Germany were found to be measly, notwithstanding the fact that in the live inspection (tongue) of the export pigs 10 per cent. were withdrawn.

Hoffmeister (von Ostertag) mentioned that in 1918 in Berlin 5 per cent. of pigs imported from Russia and the Balkan States were measly.

Berdel (1930) quotes Meyer (1929), who found that 19·47 per cent. of all slaughtered Russian bacon pigs were measly at Barnaul.

Lithuania.

At Frankfurt a.M. Berdel (1930) found, in the short period between 10th September, 1929 and 19th November, 1929, that 100 out of 1,415 imported Lithuanian pigs were measly (7 per cent.). On one day (28th October, 1929), no less than 16 out of 81 were found to be measly, and on 1st October, 1929, 12 out of 65.

Poland.

For the official abattoirs in Poland for the year 1935, Trawinski (1937) gives the following statistics:—

C. cellulosae: 12,765 cases out of 3,604,737 pigs slaughtered (0·38 per cent.).

C. bovis: 1,168 cases out of 1,148,483 bovines slaughtered (0·1 per cent.).

Sweden.

Cysticercus cellulosae is said always to have been a very rare parasite in Sweden.

According to Vosgien (1910-11), the following percentages infections were observed at the abattoir at Malmoé:—1906: 0·024 per cent.; 1907: 0·00034 per cent. (1 out of 28,616 pigs); 1908: 0·021 per cent.; 1909: 0·010 per cent.; 1910: 0·0068 per cent.

Von Ostertag supplies the following statistics for Götenburg:—1908: 0·004 per cent.; 1909: 0·009 per cent.

Denmark.

Cysticercus cellulosae has only very sporadically been found in Denmark. Thus, Vosgien states that between 1888 and 1895 only one measly pig was found out of 1,344,296 pigs slaughtered.

According to Nielsen (1934), *C. cellulosae* last appeared in Denmark before 1929.

According to Elvinge (1929), the incidence of *C. bovis* is steadily increasing in Denmark. This was very noticeable at the abattoir at Odense, between 1st January, 1927 and 1st October, 1929. In the year 1927, out of 8,483 slaughtered adult bovines, 2·07 per cent. had dead measles and 0·12 per cent. live measles. In 1928, out of 9,145 adult bovines, Elvinge found 3·15 per cent. with degenerated measles and 0·26 per cent. with live measles. In the year 1929 (9 months only) in 6,959 adult bovines, Elvinge found degenerated measles in 2·71 per cent. of carcasses and live measles in 0·39 per cent. of carcasses. The mean percentage for adult bovines was 2·90 per cent. Elvinge then gives statistics for the year 1922, in which the percentage measles in adult bovines was 0·18. The cattle originated from the same areas.

Nielsen (1934) states that *C. bovis* is increasing in some localities. At Sonderborg the incidence is 1·21 per cent. of inspected carcasses.

During the years 1920-32, out of 17,889 exported Danish bovines slaughtered at the abattoir at Basel, Switzerland, 90 were found measly (0·50 per cent.).

B. The Incidence of Cysticercosis in Swine and Bovines in Asia.

Syria.

It is not known to what extent cysticercosis occurs in Turkey proper, but it is surmised that the incidence of *C. cellulosae* must be negligible, on account of the predominant Mohammedan population. Definite statistics of the incidence of *C. bovis* are, however, available for certain Mandated States, which formerly formed part of Levantine and Asiatic Turkish Empire, e.g., Syria and Lebanon and also Palestine.

At Homs, Syria, Valade found 116 cases of cysticercosis in 615 bovine carcasses (i.e., 18·86 per cent.) in 1925-26.

Reference is made, in a subsequent part of this work, to a survey made by Yenikomshian and Berberian (1934) of the incidence of *T. saginata* infection in various parts of Syria and Lebanon. Although these authors do not give any statistics of *C. bovis* infection in cattle, it is reasonable to presume that *C. bovis* is very frequent in Syria and in parts of Lebanon, where raw beef, as " Kibbi neyyi " is customarily eaten, and the incidence of *T. saginata* is up to 12 per cent. in certain parts. The authors stress the absence of *T. solium* infection, due to the fact that in many parts of the country Mohammedanism is the predominant Faith, thus implying that *C. cellulosae* must be correspondingly rare in pigs.

Reference is also made to the survey by Penfold, Penfold and Phillips (1936), who found that more than one-quarter of the Syrian-born inhabitants of the State of Victoria, Australia, were *T. saginata* carriers. (See Part V.)

Palestine.

Mr. J. M. Smith, M.R.C.V.S., Chief Veterinary Officer to the Government of Palestine, writes (15.1.37):—

> " 1. The incidence of *Cysticercus cellulosae* is very low in Palestine, and very few swine are kept. For instance, during the last ten years only 2,112 pigs were slaughtered at the Municipal Abattoirs of Jerusalem, and of these four only were found to be affected with *Cysticercus cellulosae.*
>
> 2. With regard to *Cysticercus bovis*, this disease is endemic in this country. According to Jerusalem Abattoir figures, 10 per cent. to 22 per cent. of the cattle drawn from Hebron and Nazareth sub-districts were found to be affected with *C. bovis*. The percentage in respect of cattle drawn from other areas is lower.
>
> 3. The average annual percentage of *C. bovis* in the Jerusalem slaughter-house varies from 6 per cent. to 8 per cent."

Arabia, Iraq, Iran, Hedjaz and *Oman.*

No statistics are available for these territories, but, speculatively, one may reasonably presume that on account of the prodominantly Mohammedan populations, *C. cellulosae* must be very rare, whereas, like in Syria and Palestine, and on account of the proximity of these territories to Syria and Palestine, *C. bovis* must be a very frequent parasite.

Persia and *Afghanistan.*

No statistics are available for Persia and the more primitive Afghanistan.

Siberia.

Hjortlund, according to von Ostertag, found that 12·5 per cent. of Siberian pig-fillets were measly, when imported into Copenhagen.

Kowalesky (according to Vosgien) found at the abattoir at Tachkend (Turkestan, Russian Siberia) the following percentages of pigs measly:—1907: 0·641; 1908: 1·013; 1909: 0; 1910: 0·540.

India.

As regards the actual incidence of *C. cellulosae* in pigs in India, literature is extremely silent, and what little has appeared has frequently been somewhat contradictory. Thus, many British medical observers have stated that only the lowest caste Indians will touch or consume pork, and on that score they have presumed that the incidence of *C. cellulosae-T. solium* must be relatively low in India.

Rao (1935) mentioned the presence of *C. cellulosae* in the Madras Presidency and mentioned that he felt sure that the incidence was considerably higher than had been anticipated, so also was the incidence of *T. solium.* Then again, what is claimed to be the only recent authentic records concerning the prevalence of cysticercosis in swine and bovines, were published from Madras and Coimbatore where it was stated that 50 per cent. of swine were infected with *C. cellulosae.* (*Indian Vet. Journ., Vol.* 3, *p.* 52, 1926-27.) The same notes give the incidence of *C. bovis* in Madras and Coimbatore to be 1 per cent. Gaiger, in his check list of parasites in the Punjab, mentions the existence of bovine cysticercosis Mr. J. F. Shirlaw. M.R.C.V.S., of the Imperial Institute of Veterinary Research, Muktesar, mentions, however, in a letter dated 23rd March, 1937, that his impression, gauged on ten years' service in the Punjab, is that the disease must be of infrequent occurrence, since he found no measles in any bovines in routine post-mortem examinations.

It is astonishing that the recorded incidence of *C. bovis* should be so low in India at the present time, since during the latter part of the last century several English writers, and especially Fleming (Neumann, 1892) found in Punjab in 1869 that 5·55 per cent. of cattle slaughtered and inspected by him were heavily infected, and in 1868, 6·12 per cent.

Malaya.

At the abattoir at Singapore during 1935, Mr. J. T. Forbes, M.R.C.V.S., Municipal Veterinary Officer, found the following percentages of cysticercosis:—

Country of Origin.	Percentage in Beef.	Country of Origin.	Percentage in Pork
Bali	3·03	Bali	0·67
Siam	1·14	China	0·05
Saigon	0·85	Saigon	0·33
Malaya	1·17	Malaya	0·0004

NUMBER OF ANIMALS SLAUGHTERED IN SINGAPORE DURING 1935 AND ORIGIN.

Origin.	Cattle.	Pigs.
Bali	6,387	75,187
Siam	5,656	2,486
Saigon	1,880	37,416
Malaya	837	126,180

In a letter dated 19th November, 1936, Mr. Forbes, writes:—"Singapore depends largely on outside sources for its supply of slaughter animals. We have a very large Chinese population in Singapore, which accounts for the large number of pigs slaughtered. The Chinese usually roast their pork to a cinder and this may account for the rarity of Cysticercosis-Taeniasis in that group."

The analysis supplied by Mr. Forbes is interesting, since it shows separately the respective percentages measly animals found for the various countries from which Singapore derives its meat supply.

As regards incidence of infection of stock of purely Malayan origin, it may be noted that Mr. Forbes records that 1·17 per cent. of bovines were infected and only 0·0004 per cent. of pigs.

French Indo-China. (*Cochin China* and *Annam.*)

In Part III of this work mention is made that Bergeon (1928) frequently found *C. cellulosae* among dogs (138 cases in five years) in Hanoi, Tonkin. Bergeon mentioned the frequency of *Taenia solium* among the Tonkinese. Although the actual incidence of infection in pigs is not given, it can reasonably be presumed that *C. cellulosae* does, with frequency, occur in pigs in that territory.

Bergeon also mentioned that *T. saginata* is also readily found in Tonkin, hence, speculatively, we may attribute this frequency to a fairly high incidence of *C. bovis* in that territory.

From a point of view of territorial incidence survey, we may here repeat that Mr. J. T. Forbes, in 1935 found that 0·85 per cent. of 1,880 bovines and 0·33 per cent. of 37,416 pigs exported from Saigon, were found measly at Singapore abattoir.

Siam.

The present author was unable to obtain authentic data of infection from this country, but that *C. bovis* occurs relatively frequently in Siam, may be speculated from the report of Mr. J. T. Forbes for Singapore. In 1935, Siam exported 5,656 cattle to Singapore, of which number 1·14 per cent. were infected with *C. bovis*.

Netherlands East Indies.

At the instigation of the Dutch Colonial Government, le Coultre (1928) made a detailed enquiry into the incidence of cysticercosis in bovines and pigs on the Island of Bali in 1927. Le Coultre also had comparative statistics for some of the other parts of the Netherlands East Indies. By careful inspection, le Coultre found that on Bali 2 per cent. to 3 per cent. of pigs were infected, and 20 per cent. to 30 per cent. of bovines were measly. At Boeleleng the percentage was as high as 32·23 (407 out of 1,260). At Makassar in 1927, 1·29 per cent. of pigs were found measly, and at Soerabaia 0·6 per cent. At Denpasar in 1927, 22 per cent. of bovines were found to be measly (178 out of 809). At Mataram (Lombok) in 1926, 5·6 per cent. of bovines were measly. At Batavia in 1925, 3·26 per cent, of oxen were measly.

According to the *Tijdschrift voor Diergeneeskunde* 60, page 915, 1935, it would appear that there has been little or no decrease in the incidence of cysticercosis on Bali. The statistics given in that volume of the *Tijdschrift* are bovines 23·59 per cent. infected and pigs 2·98 per cent. infected.

Note that at Singapore in 1935 Mr. J. T. Forbes found that only 3·03 per cent. of Balinese bovines and 0·67 per cent. of pigs were measly.

China. (Including Hong Kong and Shanghai.)

As in the case of India, and indeed, of the Orient generally, statistics regarding the incidence of cysticercosis in pigs and cattle in China are most vague, and it has been almost impossible to arrive at a true estimate of the prevalence of this condition in that country.

In Shanghai and Hong Kong meat inspection is carried out under the control of European veterinarians, and in both those cities it would appear that no cases of either parasite have been found for a number of years.

Chinese medical literature occasionally quotes sporadic occurrences of *C. bovis* and/or *C. cellulosae*, but as far as is known no article has yet been published, which portrays a true reflection of the incidence of cysticercosis.

Dr. H. Pedersen, Municipal Veterinary Officer, Shanghai, writes (12.1.37):—" Uniform inspection in Shanghai over a period of many years has not revealed a case of either of these infections in hogs or bovines. It would appear thus that these parasites are non-existent in the areas from which we obtain our supplies. It is known, however, that *C. cellulosae* is prevalent in North China, but with the exception of Tsingtao, where it is stated that this infection is present amongst hogs to the extent of about 1½ per cent. we have no statistics." Similar letters were received from Messrs. D. L. McWhirter, M.R.C.V.S., and H. C. Watson, M.R.C.V.S., both of whom have had vast experience in meat inspection in the French Concession at Shanghai and at Hong Kong, respectively.

Gear and Pedersen (1934) mention that in the Shanghai municipal inspection no case of *C. bovis* was found, nor has it ever been reported from Hong Kong. Similarly, as regards *C. cellulosae* in pigs, meat inspection in Shanghai of over one million pigs did not reveal a single specimen of *C. cellulosae*; and in Hong Kong from 1910 to 1933, where an examination of over 200,000 pigs has been made annually, only two cases are reported, both in 1928.

Kuang Wu (1936) mentioned that Shu (1935) made a survey of helminths in cattle in Soochow, but he did not find *C. bovis*. In Hong Kong, Chen (1935), according to Wu, did not find *C. cellulosae* and *C. bovis* among the animals he studied; and in Canton, Chen (1936) reported the absence of *C. cellulosae* among the hogs he examined. Wu failed to find *C. cellulosae* or *C. bovis* in the abattoirs at Hangchow.

Faust (1923), according to Mills (1923), wrote: "Twenty-five years ago infestation with *T. saginata* was common in North China. The infection was brought down from beyond the Great Wall, by cattle which were slaughtered immediately and offered for sale on the markets. To-day such infection occurs rarely in Peking and vicinity. The cattle come from the same locality and are presumably infected, but for economic reasons they are fattened for a period of from several months to a year in local yards and, when slaughtered, are relatively free from infection." Mills states that these remarks by Faust are somewhat misleading, and that Faust was mistaken. Mills secured various samples of beef from a butcher in Peking. One piece, weighing three pounds, contained four measles, and another weighing five pounds, contained ten measles. By casual examination, the butcher, a German, found five infected animals in less than 300 examined, or roughly 2 per cent. Mills points out that all this meat was taken from the hind legs, therefore, according to the theory of commoner seats of infection, a far higher number would have been found if the predilection sites (head, tongue, etc.), had been carefully examined.

Mills (1924) recorded two cases of *C. cellulosae* from pigs in Peking.

Japanese Empire.

According to Prof. S. Yoshida, *C. cellulosae* in pigs has never been found in Japan proper, and *C. bovis* very rarely in cattle.

Dr. S. Yokogawa, of Formosa, however, suggests a relative prevalence of *C. cellulosae* in pigs in Manchukuo, in the fact that out of 18 cases of human cysticercosis reported from Japan proper, no less than 16 contracted the infection in Manchukuo.

According to Eguchi and Nishiyama (1930), it would appear that *C. cellulosae* is a rare parasite in pigs everywhere in Japan, except in the Prefecture Okinawa, where it is fairly prevalent. These authors supply an interesting table showing the incidence of *C. cellulosae* in this Prefecture, from their observations at abattoirs, and they found that in 1916 only 0·01 per cent. of pigs were infected. In 1920, infection was 0·91 per cent. and in 1923, 1·03 per cent., which percentage remained more or less uniform until 1926, when there was a sudden rise to 2·71 per cent., and thereafter a steady decrease, illustrated thus: 1927: 2·14 per cent.; 1928: 1·83 per cent.; 1929: 0·94 per cent.

Prof. Yoshida supplies a recent translated article by Nakanishi, who found that in Korea 33 per cent. of adult cattle were measly. Nakanishi (1926) found that 37·5 per cent. of Korean calves were measly.

C. THE INCIDENCE OF CYSTICERCOSIS IN SWINE AND BOVINES IN OCEANIA.

Australia.

According to Drabble (1934), *Cysticercus cellulosae* has never been found in Australian pigs.

A mild outbreak of *C. bovis* was recorded from the State of Victoria a few years ago. This outbreak occurred as the result of the grazing of slaughter cattle on the Werribee Sewage Farm, and caused great consternation among the meat-consuming public of Victoria.

According to personal advice from Mr. Drabble, a few (not more than half a dozen) sporadic cases of *C. bovis* have been found on meat inspection in abattoirs in New South Wales over a number of years.

New Zealand.

Mr. W. C. Barry, M.R.C.V.S., Director Live Stock Division, Department of Agriculture, New Zealand, writes (5.1.37):—

> "So far as is known, no cases of cysticercosis in pigs or cattle have occurred in this Dominion at any time."

Phillipine Islands.

Schwartz and Tubangui (1922) obtained statistics from the Ascarraga abattoir, Manila, which showed that just over 1 per cent. of pigs were infected with *C. cellulosae*. This was the average over five years.

C. bovis is rarely found in the abattoir at Pandacan, Manila (Schwartz, 1925), but native cattle are never slaughtered at abattoirs and their meat is thus never inspected. The incidence of *T. saginata* is considerably higher than that of *T. solium*. (Schwartz and Tubangui, 1922.)

D. THE INCIDENCE OF CYSTICERCOSIS IN SWINE AND BOVINES IN THE AMERICAS.

Canada.

According to Mr. George Hilton, Veterinary Director-General, Canada, measles in pigs and cattle is a very rare disease in Canada.

The Report of the Veterinary Director-General for the year ended 31st March, 1935, gives the following statistics:—

Cysticercus bovis found at establishments under inspection:
44 Carcasses. (Bovine.)
774 (Portions) of carcasses. (Presumably infection was confined to heads, viscera, etc.)

Cysticercus cellulosae.
42 Pig carcasses.
12 (Portions) of carcasses. (Presumably infection was confined to heads, viscera, etc.)

During the year under report 1,350,370 bovines were slaughtered in Canada, and 2,862,125 pigs were slaughtered. Presuming that each of the measly "portions" came from separate measly animals, 818 measly bovines and 54 measly pigs were found during that year, reflecting a very low percentage.

It is, however, interesting to recall that during the years 1920-32 Dr. Unger found 16 out of 4,652 bovines of Canadian origin to be measly at the abattoir at Basel in Switzerland, representing a percentage of ·34.

United States.

In submitting a tabulated statement showing the numbers of each species of animal slaughtered, by years, from 1926 to 1935, in which the number of carcasses of each species condemned on account of cysticercosis is given, Dr. J. R. Mohler, Chief of the Bureau of Animal Industry, United States Department of Agriculture, writes (2.11.36):—"Inasmuch as all infested carcasses are not condemned on account of slight cases of infestation being passed after prescribed freezing or sterilization, this does not supply information upon which percentages at which the condition prevails may be determined."

Year.	Cattle.		Swine.	
	Slaughtered.	Condemned.	Slaughtered.	Condemned.
1926	10,098,121	129	40,442,730	76
1927	10,049,589	169	42,650,443	71
1928	9,040,028	121	48,347,393	57
1929	8,284,324	123	47,163,573	61
1930	8,280,778	131	46,688,860	98
1931	8,215,203	99	44,047,458	58
1932	7,974,502	103	45,852,422	21
1933	7,735,588	125	45,698,053	20
1934	9,652,952	149	45,773,196	35
1935	12,809,448	257	34,413,317	38

Since the "Lightly" infested carcasses are not given in Dr. Mohler's summary, we may, according to general observations presume that at least ten times the number of measly carcasses shown were treated by freezing, etc. Multiplying thus the number by ten, 2,570 measly carcasses out of approximately 12,000,000 were found in 1935, or roughly 1 in 5,000—still a very low incidence.

According to Ransom (1911), the average percentage of *C. bovis* at that time was 0·6. Later (1913), Ransom stated that 1 per cent. of all cattle slaughtered in the United States were infected with *C. bovis* (*Journ. of Agric. Research*, *Vol.* 1, *p.* 15).

According to Price (1925), *C. cellulosae* is frequently found in Texas in pigs. Price points out that this is understandable considering the large Mexican and Negro populations.

Central America.

According to Hall (1927), the incidence of *C. cellulosae* in swine in Central America is astonishingly high, the parasite occurring in from 5 to about 30 per cent. of swine, usually in gross infestations. As a result of rigid sanitation caused by a campaign against hookworm in Panama, Dr. Mattatall, according to Hall, reported that at the Panama City abattoir the incidence of *C. cellulosae* dropped from 15 per cent. to 5 per cent. According to Hall " the occurrence of *T. saginata* in man in the Central American countries shows the concomitant presence of *C. bovis* in cattle. Dr. Mattatall, however, finds the *C. bovis* to be a very rare parasite in Panama City."

Nauck (1931) wrote that *C. cellulosae* was a common disease in Costa Rica.

West Indies.

According to Cameron (1930), *C. bovis* is occasionally found in the West Indies. *C. cellulosae* is sometimes seen, most frequently in the Southern Islands.

Brazil.

No definite data have been obtained from Brazil, but Palais (1933) refers to the occurrence of *T. saginata*, which would suggest a corresponding frequency of *C. bovis*.

Argentine.

The incidence of *C. bovis* and *C. cellulosae* is relatively low in the Argentine, as is shown by the subjoined table forwarded by Señor A. Andrieu, Chief of the Sanitary Police, Buenos Aires.

The figures show the number of cases and the numbers per 10,000, as observed at the principal abattoirs and *frigoríficos* during the five years 1932-1936:—

Year.	Bovines. (*C. bovis.*)		Pigs. (*C. cellulosae.*)	
	Cases.	No. per/10,000.	Cases.	No. per/10,000.
1932	204	0·92	11	0·25
1933	461	1·97	67	1·04
1934	1,322	5·06	343	3·77
1935	2,254	8·50	342	3·74
1936	1,671	5·48	860	8·71
TOTAL	5,912	4·58	1,623	4·28

Chile.

Señor Rogelio Montero, Chief of the Meat and Animal Sanitation Department, Santiago, supplies the following statistics showing the incidence of *C. cellulosae* in swine as observed at the Santiago abattoir.

Year.	Condemned.	Total Inspected.	Percentage Measly.
1933	3,242	77,199	4·2
1934	3,410	89,042	3·8
1935	3,880	98,653	3·9
1936	3,493	92,862	3·8

Señor Montero states that no statistics are available of the incidence of *C. bovis* in Chile, but this is quite an uncommon disease.

E. THE INCIDENCE OF CYSTICERCOSIS IN SWINE AND BOVINES IN AFRICA.

Tunis.

At the abattoir at Sousse, Coussi (1933) found the incidence of *C. bovis* (average for five years) to be 2·25 per cent.

According to some of the older writers, e.g., Alix (1887), it was formerly estimated that 5 per cent. of bovines in Tunis were infected with *C. bovis.*

Senegal.

At Dakar, Teppaz (1923) estimated the incidence of *C. bovis* at approximately 10 per cent.

French Guinea.

Claverie (1928) found that approximately 50 per cent. of bovines were infected with *C. bovis* in French Guinea.

Sierra Leone.

Mr. J. Martin, Director of Agriculture, Sierra Leone, supplies the following data in respect of the incidence of *Cysticercus bovis* as observed at the abattoir at Freetown. Mr. Martin states (letter dated 1st February, 1937), that there are no other centres in Sierra Leone in which cattle are slaughtered to any extent:—

Year.	Bullocks slaughtered.	Measly.	Percentage.
1931	2,818	2	0·0709
1932	2,904	1	0·0343
1933	4,593	20	0·435
1934	4,460	18	01403
1935	4,274	6	0·140
1936	3,278	10	0·305
TOTAL	22,327	57	0·255

It may here be mentioned that Maplestone (1924) found 3·32 per cent. of 500 inmates of Freetown gaol to be infected with *T. saginata.*

Abyssinia.

It is not known to what extent infection with *T. saginata* occurs among Abyssinians at the present time, or what the present incidence of *C. bovis* is in that country, but about forty years ago, according to several writers (Leuckart, Neumann, von Ostertag, etc.), practically 100 per cent. of the Abyssinian population considered " a *Taenia saginata* one of their most treasured possessions," and correspondingly, it is presumed that a very big percentage of bovines must have been measly.

Kenya Colony.

Cysticercus cellulosae is a relatively uncommon parasite in pigs in Kenya, but it does occur sporadically. For instance, in Nairobi abattoir in 1934, four pigs were condemned out of 1,959 pigs inspected, whereas in 1935, *C. cellulosae* was not detected at Nairobi abattoir, " but one case of extremely heavy infestation was diagnosed at the Veterinary Research Laboratory." (Daubney, 1936.)

A steady increase in the incidence of *C. bovis*, as observed in the Nairobi abattoir, is reflected in the subjoined table.

The Medical Officer of Health, Nairobi, recently informed Stock Owners' Conference that were the standard raised so that any animal with a single viable *Cysticercus* was condemned, the percentage of condemned cattle would be increased by 4·7 in the case of grade cattle and by 7·4 in the case of native cattle. (NOTE.—Cattle are not condemned unless six viable measles can be demonstrated in the carcass.) If all measly cattle were thus to be condemned at Nairobi the incidence of *C. bovis* would be in the vicinity of 25 per cent.

Table from the Seventh Annual Report of the Medical Officer of Health, Nairobi.

OXEN SLAUGHTERED AND CONDEMNED FOR MEASLES.

Year.	Grade.			Native.			Total.		
	Killed.	Condemned.	Percentage condemned.	Killed.	Condemned.	Percentage condemned.	Killed.	Condemned.	Percentage condemned.
1927	5,634	—	—	5,178	—	—	10,812	490	4·5
1928	4,907	—	—	6,827	—	—	11,734	740	6·3
1929	4,151	—	—	7,617	—	—	11,768	975	8·2
1930	4,214	277	6·5	7,243	683	9·4	11,457	960	8·3
1931	4,306	388	9·0	9,375	1,227	13·0	13,681	1,615	11·8
1932	3,054	321	10·5	11,044	1,568	14·1	14,098	1,889	13·3
1933	2,924	326	11·1	12,968	2,158	16·6	15,892	2,484	15·6
1934	4,531	600	13·2	10,264	1,820	17·7	14,795	2,420	16·3
1935	4,806	495	10·2	9,007	1,894	21·0	13,813	2.389	17·2

Uganda.

The Acting Director of Veterinary Services gives the following statistics reference to the incidence of *C. bovis* at the Kampala abattoir for 1935:—

Cattle Slaughtered	4,336
Condemnations—	
Hearts	685
Tongues	248
Quarters	140
Complete Carcasses	58

It is difficult to understand these figures, but on the presumption that measles were found in 685 ox hearts (ignoring the tongues, quarters and carcasses), then 685 out of 4,336 bovines were measly, or 15·8 per cent. Including the possible number of bovines in which measles may only have been found in the tongue, or in a quarter, or in a carcass, and not in the heart, it can be concluded that from 15 per cent. to 25 per cent. of the Uganda cattle are infected with *C. bovis*. The Director states that the percentage of infected carcasses amongst Western Province cattle is higher than amongst Eastern Province stock, both areas might be termed "Native Reserves" as there are no European owned stock farms in either area.

Tanganyika.

In 1916 von Ostertag referred to the wide distribution of *C. bovis* in both British and German East Africa, before the war. He also mentioned the frequency of *T. saginata* infection in those territories among natives, owing to their habits of eating imperfectly cooked meat. Von Ostertag quoted Veterinary Officer Manleitner, who found a very high percentage infection in cattle in Aruscha; Veterinary Officer Meyer, who found 2 bovines out of 14 measly in Shirati; whereas in Muansa Veterinary Officer Gärtner found no measles in 24 bovines examined. In Bukoba, von Ostertag estimated that 90 per cent. of bovines were infected. In general, infection ranged from 1 to 10 per cent. or higher.

At that time (about 1916) no definite survey had been made of the incidence of *C. cellulosae* in pigs, but von Ostertag mentioned that most of the pigs consumed before the war were imported from the Union of South Africa, where the percentage infection in pigs was said to be very high, according to von Ostertag.

Hammer (1922) states that during his period of service in German East Africa he found approximately 15 per cent. of bovines measly in the Uhehe Highlands. Hammer's pre-war findings coincided very nearly with present day statistics from Tanganyika.

Captain H. J. Lowe, M.R.C.V.S., Veterinary Research Officer, Mpwapwa, supplies the following tables showing the monthly percentages measles found at various abattoirs from January to August, 1936. Only when more than 2 per cent. of cases were found, were these included in the returns. According to Capt. Lowe, practically all the beef consumed in the Territory is derived from native-owned animals, that is from Native Reserves.

During the period under report only one pig was condemned (during May, at Iringa), and a total of 392 pigs were slaughtered at all abattoirs.

Tanganyika Territory.—Abattoirs at which more than 2 per cent. of Bovine Carcasses were Sterilized for C. bovis.

Abattoirs.	1936 : Percentages.							
	Jan.	Feb.	Mar.	April.	May.	June.	July.	Aug.
Tukuyu	16·1	—	6·6	—	—	—	2·8	9·1
Iringa	14·9	15·1	15·5	12·6	21·6	8·1	11·7	14·3
Moshi	9·4	7·4	8·0	6·6	9·1	13·5	11·8	7·0
Dar-es-Salaam	9·1	12·4	13·2	13·0	10·8	8·8	9·3	9·2
Morogoro	8·5	8·3	6·0	9·3	—	3·8	3·4	3·4
Kondoa	6·9	—	—	—	—	11·1	6·2	—
Aruscha	6·5	—	6·2	6·6	8·8	—	2·5	3·6
Singida	4·3	6·2	5·2	11·1	6·6	—	—	3·8
Dodoma	3·4	4·6	4·7	6·1	6·1	4·6	6·6	9·1
Korogwe	2·5	—	—	3·2	—	3·0	—	—
Mpwapwa	—	16·6	14·3	18·4	11·1	18·6	3·2	3·6
Musoma	—	3·3	4·5	10·5	3·4	8·6	—	8·0
Mbeya	—	—	5·4	2·3	5·5	8·9	9·8	13·3
Songea	—	—	—	7·4	—	4·0	—	5·3
Tanga	—	—	—	—	—	2·3	—	3·4

Judging from these monthly returns, it would appear that Iringa, Moshi, Dar-es-Salaam and Mpwapwa draw their slaughter cattle from the centres of heaviest infection.

Belgian Congo.

Prof. Rubray, Rector of the Royal College of Veterinary Medicine at Cureghem-lez-Bruxelles, Belgium, kindly supplied the following statistics, relative to the incidence of *C. bovis*, as observed at the abattoir at Stanleyville, from January to October, 1936:—

	Bovines Slaughtered.	Infested with Cysticercosis.	Percentage Infested.
January	69	6	8·7
February	72	5	6·9
March	78	4	5·1
April	75	7	9·3
May	75	11	14·7
June	70	6	8·6
July	63	10	15·9
August	68	4	5·9
September	68	5	7·4
October	72	4	5·6
TOTAL FOR TEN MONTHS	710	62	8·7

Angola.

Statistics of the actual incidence of infection with either parasite in pigs and cattle, respectively, are not available for Angola, but it is interesting to record that at Lisbon in 1933, 1934 and 1935 only 153 bovines were found measly, and of this number 151 were imported from Angola. The total number of bovines exported from Angola and slaughtered at Lisbon during those years was not given.

Madagascar.

Detailed statistics from this French African Island Colony have from time to time been published, and from these data it has been possible to compare relatively early and recent percentages of *C. cellulosae* and *C. bovis.*

Geoffroy (1906) stated that the percentage of measly pigs found at Tananarive in 1905 was 7·01.

Poisson (1926), and also at the Pan-African Agricultural and Veterinary Conference at Pretoria in 1929, stated that in Madagascar the pig is especially reared by the people of the centre of the island (*horas* and *betsileos*). At about that time the incidence of *C. cellulosae* in pigs, as observed at various abattoirs and meat canning factories was:—

At Diego-Suarez	4-5 per cent.
At Tamatave	10 per cent.
At Tananarive	12-15 per cent.
At Antsirabe	12-20 per cent.

In the districts of the bramble fields in the high plateaux, it was said to have been even higher.

Buck, Lamberton and Randriambeloma (1935) found that 13 per cent. of 4,500 pigs examined at the abattoir at Tananarive, that year, were measly.

A geographical map giving a survey of the incidence of porcine *C. cellulosae* on the Island in 1928, was kindly donated by Dr. H. Poisson, Retired Veterinary-Director-General, now domiciled in Tananarive. This map gives the following percentages:—Diego-Suarez, 2·40; Tamatave, 10·41; Tananarive, 9·76; Antisirabe, 12; Ambositra, 12; Ambohimahasoa, 12; Fianarantsoa, 13; Tulear, 2; Majunga, 6-7.

An extract of the Archives of the Veterinary Service (kindly supplied by Dr. Poisson) gives the following percentages of *C. cellulosae* as observed in Madagascar in 1936:—

(1) *Central Region*:—

Tananarive: 9·47 at Municipal abattoirs.
7·10 at Androrosy and Ambohimanarina.
11·84 at factory at Soanierana.
Antsirabe: 12·25 at municipal abattoir and factories.
Fianirantsoa: 21·20.

(2) *Eastern Region*:—

Tamatave: 7·35. (Mean average.)

Factory of Society Rochefortaise: 8·82. (Come from Centre and taken to Tamatave.)

City Abattoir: 5·89. (Come from Centre and taken to Tamatave.)

(3) *North-west Region*:—

Majunga: 6·92.

(4) *Northern Region*:—

Diego-Suarez (abattoir and factory): 3·03.

According to Poisson (1928), between 1912 and 1927 only occasional sporadic cases of *C. bovis* were observed at the various abattoirs in Madagascar. Dureiux (1934), stated that *C. bovis* had been found at abattoirs on the Island since 1917, but the maximum percentage is 3 per cent. In 1936, according to an extract from the Archives of the Veterinary Service, 0·19 per cent. of bovines slaughtered at the abattoir at Tananarive were measly.

Portuguese East Africa.

According to Dr. Jose Botelho, Abattoir Inspector and Municipal Veterinary Officer, Lourenco Marques, the average percentages during the last three years have been:—

Cysticercus cellulosae in pigs	3·6
Cysticercus bovis in cattle	3·15

Northern Rhodesia.

The Medical Officer of Health, Ndola, kindly furnished the following statistics showing the number of cases and percentages of *C. cellulosae* and *C. bovis* observed at the Ndola abattoir during the years 1932 to 1935:—

Year.	Pigs slaughtered.	*C. cellulosae* Pigs infected.	Percentage.	Cattle slaughtered.	*C. bovis* Cattle measly.	Percentage.
1932.........	122	7	5·7	1,164	65	4·7
1933.........	250	21	8·4	1,217	49	4·0
1934.........	335	17	3·1	1,652	18	1·0
1935.........	493	23	4·6	1,961	22	1·1
TOTAL.....	1,200	68	5·0 (approx.)	5,994	154	2·7

The Medical Officer of Health attributes the reduction in the percentage of infested animals to the fact that butchers now purchase slaughter stock from ranches having a "clean" record, and as far as possible avoid the purchase of animals from ranches which are known to be *foci* of infestation.

Southern Rhodesia.

It has been very difficult to obtain reliable statistics of the incidence of cysticercosis from Southern Rhodesia, since few of the townships, with the exception of Salisbury and Bulawayo have properly controlled abattoirs in which authentic statistics are kept.

The Abattoir Superintendent, Salisbury, informs me that the average annual percentage measles in pigs is about 3 per cent., and in bovines about 2 per cent.

The Superintendent of the Municipal Abattoirs, Bulawayo, has forwarded the following data, relative to observations at Bulawayo for the last five years ended 30th June, 1936:—

Swine:

(1) Number of measly carcasses: 1,434.
(2) Percentage these figures represent: 6·7.
(3) It is estimated that 1,148 (or 80 per cent.) of these measly pigs are of native origin.

Bovines:

(1) Number of measly carcasses: 230.
(2) Percentage these figures represent: 0·38.
(3) It is estimated that 180 (80 per cent.) of these measly bovines are of native origin.

Judging from the available statistics from Southern Rhodesia, it would appear that *C. bovis* is not a common parasite in that country. This may be attributed to the fact that probably a large percentage of slaughter stock, even of native origin, may be raised under semi-ranging conditions, under which they do not come in contact with humans.

South West Africa.

Windhoek is the only centre in South West Africa from which any statistics could be obtained. According to these statistics, in 1931, 1,100 pigs were slaughtered at the abattoir, of which number only one was found infected with *C. cellulosae.* Since then no cases have been found. (NOTE.—Von Ostertag in 1916 alleged that before the war it was frequently found that 50 per cent. of the pigs exported from the Cape to German South West Africa were measly. This high percentage almost trebles those from the centres showing the extreme maximum incidence at the present time. It will be noticed that but a few Transvaal and Orange Free State centres give a return of over 10 per cent. infection in pigs, so that von Ostertag's estimate appears almost fantastic.)

With reference to the occurrence of *C. bovis*, the following data are given:—

In 1933, of 3,816 bovines slaughtered, 10 were infected.
In 1934, of 3,821 bovines slaughtered, 12 were infected.
In 1935, of 3,874 bovines slaughtered, 8 were infected.
In 1936, of 2,687 bovines slaughtered in 9 months 8 were infected.

The remarkably low incidence of *C. bovis* in South West Africa may be explained on similar lines to that of Southern Rhodesia. In the next survey (that of Bechuanaland Protectorate), it will be observed that Mr. Hay found no cases of *C. bovis* among cattle from Ngamiland and Ghanzi, areas comprising vast open ranges, remote from human habitations, and bordering on South West Africa.

It will be recalled that Dr. Unger found 3 out of 321 cattle imported from South West Africa measly at Basel (Switzerland) in 1923 and 1924 (0·94 per cent.).

Bechuanaland Protectorate.

The only abattoir in this Territory is situated at Lobatsi, from which centre export beef is forwarded. Mr. W. Hay, Government Veterinary Officer in charge of meat inspection at this abattoir states that no pigs are slaughtered there, and that 1·05 per cent. are found to be measly. No measly cattle have been found among those originating from Ngamiland and Ghanzi. Commenting upon Mr. Hay's report, the Chief Veterinary Officer of the Bechuanaland Protectorate, writes (24.11.36):—" Our experience at Lobatsi shows that measles is not evenly distributed but occurs in batches of cattle, which fact has led to the reasoning that only cattle in areas thickly populated by natives contract measles."

Basutoland.

The Principal Veterinary Officer, Basutoland, writes (letter dated 30.10.36):—" It is impossible to estimate even the approximate number of cases met with on post-mortem examination throughout the Territory. The Territory is occupied by natives only and pigs are raised exclusively for domestic purposes. Pigs are either kept in sties or allowed to range. The latter virtually become village scavengers and about 10 per cent. of these are infected, whereas 2 per cent. of the former are infected. In connection with cattle, I am afraid I am unable to furnish any data because we have no meat export trade."

Union of South Africa.

The statistics given in the following tables, showing the percentages of *C. cellulosae* and *C. bovis* at the various abattoirs in the Union, were obtained as the result of a personal questionnaire to the respective Abattoir Directors or Superintendents, Medical Officers of Health, Health Inspectors, or Town Clerks of the centres, all of whom kindly supplied the data given. There are, unfortunately, some centres from which most evasive replies were obtained, and a few, including fairly large towns, from which no replies whatsoever were obtained. Consequently, since it was my policy to include only first-hand authentic information in this survey, reference to the subjoined tables and the " incidence maps " will show the exclusion of some very important centres bordering on, or close to, Native Territories. If suitable statistics had been kept at these excluded centres, it is possible that some very interesting information may have been presented.

Cape Province.

Abattoirs.	Average number of *C. cellulosae*.	Average number of *C. bovis*.	Percentage *C. cellulosae*.	Percentage *C. bovis*.	Average Years.
Aliwal North........	4	14	0·5	1·5	1
Beaufort West......	1	1	0·67	0·25	3
Bedford............	—	—	2·75	1·5	5
Burghersdorp........	—	4	—	0·87	1
Cradock............	11	5	2·49	0·75	5
Capetown...........	292	492	4·26	1·12	5
East London........	438	408	7·69	5·69	3
Fort Beaufort.......	8	33	9·29	6·1	6
George.............	11	3	2·61	0·53	6
Graaff-Reinet........	19	—	3·33	—	4
Kimberley..........	59	88	1·68	1·22	10
Kingwilliamstown....	82	92	4·7	5·2	6
Mafeking...........	23	41	6·67	2·67	3
Malmesbury.........	16	2	4·27	0·32	5
Middelburg..........	14	23	2·9	0·94	5
Mossel Bay.........	1	1	1·37	—	1
Paarl...............	—	—	3·33	1·75	—
Port Elizabeth......	170	653	1·76	7·29	5
Queenstown.........	50	11	3·3	0·67	5
Riversdale..........	13	7	5·8	3·0	1
Stellenbosch.........	7	29	1·58	2·44	4
Swellendam.........	22	24	3·0	4·0	—
Uitenhage...........	20	10	3·0	0·6	5
Upington...........	—	5	—	0·89	1
Vryburg............	4	2	7·0	0·3	5
Worcester...........	11	4	1·97	0·31	5

Natal

Abattoir.	Average number of *C. cellulosae*.	Average number of *C. bovis*.	Percentage *C. cellulosae*.	Percentage *C. bovis*.	Average Years.
Dundee.............	7	62	2·70	5·80	5
Durban.............	998	928	5·16	2·68	10
Greytown...........	3	27	1·69	3·84	5
Ladysmith..........	—	—	2·0	4·0	—
Newcastle...........	20	7	2·45	0·158	—
Pietermaritzburg.....	43	477	1·77	5·3	5
Vryheid.............	8	59	2·9	4·3	10

Orange Free State.

Abattoir.	Average number of *C. cellulosae.*	Average number of *C. bovis.*	Percentage *C. cellulosae.*	Percentage *C. bovis.*	Average Years.
Bethlehem..........	32	38	15·2	2·13	5
Bloemfontein........	74	443	2·13	4·87	2
Brandfort...........	1	12	—	5·0	—
Clocolan............	9	8	9·0	2·01	3
Fauresmith.........	—	3	—	—	2
Ficksburg...........	135	7	25·0	1·09	10
Frankfort...........	6	12	5·03	2·01	—
Harrismith..........	9	—	4·51	—	3
Heilbron............	1	1	0·88	0·08	3
Kroonstad..........	41	10	4·262	0·45	5
Lindley............	6	5	6·0	2·10	2
Parys..............	13	11	5·5	1·51	4
Senekal............	21	12	25·07	2·0	10
Wepener...........	3	—	9·43	—	5
Winburg......... ...	8	1	4·44	0·3125	8

Transvaal

Abattoir.	Average number of *C. cellulosae.*	Average number of *C. bovis.*	Percentage *C. cellulosae.*	Percentage *C. bovis.*	Average Years.
Barberton..........	—	—	—	5·31	—
Boksburg...........	—	81	—	1·22	2½
Brakpan............	77	69	4·27	0·71	3
Germiston..........	32	260	1·04	1·48	5
Johannesburg.......	3,148	834	4·42	0·75	11
Klerksdorp..........	30	24	4·91	1·37	1
Krugersdorp........	80	190	6·10	1·46	4
Lichtenburg.........	5	2	19·48	0·18	3
Middelburg..........	30	30	11·49	3·04	8
Nolspruit...........	10	43	6·41	2·03	1
Nigel...............	43	90	5·02	2·80	1
Pietersburg.........	219	68	4·95	2·97	3
Potchefstroom.......	111	41	15·30	1·23	5
Pretoria............	595	297	7·85	1·98	11
Randfontein.........	31	198	3·96	2·30	4
Rustenburg.........	53	98	10·12	5·11	4
Springs.............	25	146	3·91	1·20	5
Volksrust...........	12	1	5·0	1·06	1
Witbank............	20	80	8·97	2·75	6

Average Number of Carcasses per Year.

Cape Province	1,276	*C. cellulosae.*	1,952	*C. bovis.*
Natal	1,079	,,	1,560	,,
Orange Free State ...	359	,,	563	,,
Transvaal	4,521	,,	2,551	,,
TOTAL FOR UNION	7,235	,,	6,626	,,

Discussion.

In a note which was compiled by Dr. H. H. Curson towards the end of 1936, for a Native Affairs Departmental Bulletin, and accompanying which two tables and graphs were supplied, the position in the Union is very clearly defined. The statistics given in the tables of Dr. Curson's note, are subjoined hereto, and are in respect of the nine principal abattoirs in the Union, plus that of Kingwilliamstown, which town borders on the Transkeian Territories. Reference to the graphs shows a steady increase in the numbers and percentages of measly bovines and pigs, from observations at the respective abattoirs. Undoubtedly the steady increase in the incidence may be due to general better inspection technique, but also, it may be possible that a larger percentage of slaughter stock is derived from native areas.

The accompanying " Incidence Maps " may not be quite indicative of the actual incidence of infection in the various areas. For instance, no details were obtainable from abattoirs in, or close to definite native areas, such as Eshowe, Kokstad, Umtata, Grahamstown, Kuruman, Zeerust, Waterberg, Lydenburg, or Zoutpansberg. Yet, the abattoirs at Durban, East London, Port Elizabeth and Johannesburg obtain a fairly large percentage of their slaughter pigs and cattle from those areas. For smaller centres, the figures and percentages may be accepted as almost truly indicative, since stock slaughtered at the smaller abattoirs are generally reared in the same districts. At the Bloemfontein abattoir we were able to trace definite " black " areas of origin during the past three years. Thus, in consignments from Theunissen in the Orange Free State, and also from Thaba 'Nchu and Tweespruit, we frequently found a fairly large percentage of infected cases.

Mr. W. A. Dykins, M.R.C.V.S., the author's colleague in Durban reports (letter dated 21st September, 1936) " the incidence of measles in cattle in the years under review has increased, and regarding pigs the converse seems to be the case. I do not think any special significance should be attached to the latter, as farmers and others who have doubts about their pigs do not consign them to abattoirs where efficient meat inspection is in vogue, so, in my opinion, the low incidence gives rise to a wrong impression ". Mr. Dykins adds that the cattle with the highest infection come from native areas such as Swaziland, Gollel, Candover, Mkuzi, Richmond and Ixopo, and attributes this, naturally, to " absence of proper sanitary measures ".

In another letter, dated 20.6.36, Mr. Dykins stated: " I definitely find that the highest percentages of measles are to be found amongst cattle ex native areas, such as Swaziland and the portions of Zululand contiguous thereto. A high percentage is frequently met in animals from the Midlands of Natal, and actually from the so-called well managed farms ".

The Town Clerk, Newcastle, writes (17.11.36): " The greatest number of cases at this abattoir have been in pigs and cattle purchased in or near the Utrecht (Natal) District. As regards percentage infection at East London, Dr. P. W. Laidler, Medical Officer of Health, writes (11.1.37): " A large proportion of the stock was from native areas ".

Mr. H. J. Lubbe, Abattoir Superintendent, Graaff-Reinet, writes (19.11.36): " We have had no records of measles in bovines at this abattoir. As regards the origin of measles in swine, farmers in this District allow their swine to run wild amongst the prickly pears, which we have here in abundance. Sanitary conveniences are provided on most farms for the Europeans only, the natives being allowed to use the veld ".

Mr. H. A. Waterson, Health Inspector, Mafeking, writes (30.12.36): " The percentage infection in pigs was very high in 1933 and 1934, as most pigs slaughtered at that time were brought from Native Reserves ". (The percentages given for 1933 and 1934 were 7 and 8, respectively.)

Mr. C. J. Grobler, Health Inspector, Malmesbury, writes (27.10.36): " It will be observed that the incidence of measles in bovines is comparatively low. This is due to the fact that, as a country town, local butchers must of necessity slaughter from a reserve, that is, selected stock and not direct from rail or the open market as in large centres. In purchasing stock, butchers steer clear of coloured areas and natives territories, for instance Queenstown and vicinity. Local supplies of bovines are very limited and are obtained from as far afield as Okanja and Gobabis in South-West Africa, from the Eastern Province and from Namaqualand; consequently the recorded incidence of *C.bovis* at this abattoir cannot be taken as a criterion for the Malmesbury area, where it is of very rare and doubtful occurence, while pigs are bought and raised purely locally, and *C.cellulosae* is fairly rife".

Mr. J. L. Marais, Health Officer, Middelburg, Cape, writes (12.12.36): " Most cases of measles found here, during the past five years have been in oxen from the Transkei. No cases of measles have ever been found here in cattle bred in the Middelburg District".

Mr. D. Benham, Health Inspector, Riversdale, writes (9.11.36): " All the oxen infected came from the same part of the district, and since the butchers have stopped buying from that area, I have not found any measly carcasses ".

Mr. L. Becker, Abattoir Superintendent, Swellendam, writes (24.11.36): " Pigs coming from areas exclusively or predominantly inhabited by coloured people, or from farms along the main arterial roads, are obviously treated with suspicion, even by the butchers of towns of the size of Swellendam ".

Mr. G. P. Louw, Health Inspector, Upington, writes (31.10.36): " In the last ten months, one case of *C. bovis* was definitely of native origin, four others came from South-West Africa ".

Mr. C. M. de Jager, Abattoir Superitendent, Volksrust, writes (26.10.36): " The majority of bovine cases of measles are animals purchased from natives, sepecially from the Lowveld ".

Mr. E. J. Scallan, Health Inspector, Rustenburg, writes (27.10.36): " We have farmers in the district who speculate in cattle and pigs and purchase these animals from natives and sell them on the sales or to the butchers as their own ".

Mr. F. R. Carter, Abattoir Superintendent, Potchefstroom, writes (30.11.36): " About 15 per cent. of cattle slaughtered here are of native origin, and about 75 per cent. of cattle bought from native areas are condemned. Most of the pigs slaughtered at this abattoir are drawn from native areas ". Mr. Carter stated that when he first arrived at Potchefstroom 10 years ago, the highest condemnation of meat was 5,000 lb. weight. During the first 8 months of his service he condemned 40,000 lb. weight, and nowadays the condemnation weights are less than half that amount. Mr. Carter states that " the butchers are now very careful where they buy their stock".

The Abattoir Superintendent, Nigel, states (28.10.36) that the majority of pigs slaughtered at that abattoir are obtained from farmers in the locality. It has been his experience, however, that the majority of pigs of known native origin have been infected with measles.

Mr. D. Arnold, Abattoir Superintendent, Krugersdorp, writes (10.11.36): " Oxen slaughtered here are bought all over the country, but the principal sources of supply are the Johannesburg Market, parts of the O.F.S., and Rustenburg. From Rustenburg we get about 30 per cent. oxen per month and to my mind about 60 per cent. would be of native origin ".

The Town Clerk, Barberton, writes (27.10.36): " In such centres as Sabie, Noordkaap, Sheba, Eureka, Louwscreek, Hectorspruit, Komatipoort, Kaapsche Hoop, Nelshoogte, animals are slaughtered in abattoirs where no post-mortem examinations are made. It has been conclusively proved in the Barberton Municipal Abattoir, that the incidence of measles (*cysticerci*) in cattle is on the increase. During the past six months, of all those slaughtered, the percentage infested was as high as 5·31 ".

Mr. P. G. Joubert, Health and Meat Inspector, Fauresmith, makes the following observation in regard to the origin of infected bovines at various abattoirs where he formerly served in the Cape, (letter dated 29.10.36): " Much depended on the vicinity from which stock were obtained. For instance, it was noticed that bovines from the Eastern Province were the most frequently infested, with the Transvaal a good second and the Free State third. Measles disease was practically never found in stock brought from South-West Africa. Measles was common in pigs reared at the Cape ".

In his Annual Report for the year ended 30.6.35, Col. J. Irvine-Smith, Director of Abattoir Department, Johannesburg, makes the following observation. (Page 3): " Measles infestation (bladder-worm) of export cattle from Natal ranges from 2·7 per cent. to 60 per cent., with an average of 4·05 per cent.

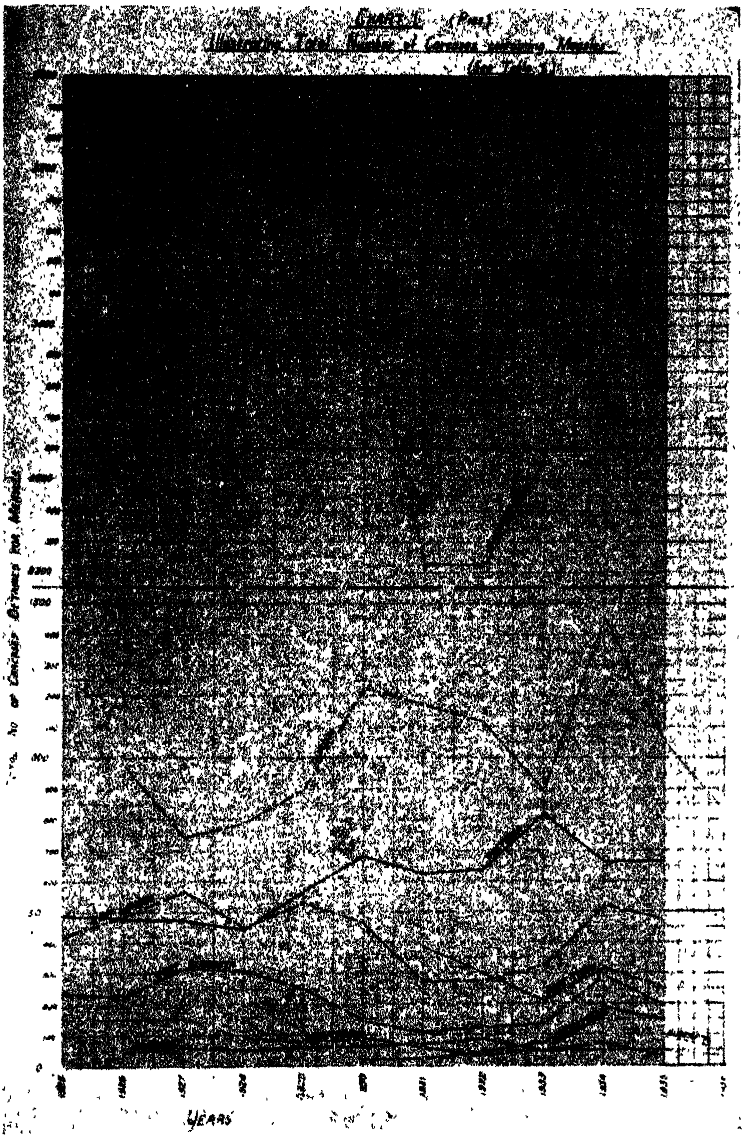

CHART I (PIGS).

Graphical Illustration showing the Total Number of Carcasses containing Measles at the nine Principal Abattoirs of the Union, plus Kingwilliamstown, adjoining Transkei.

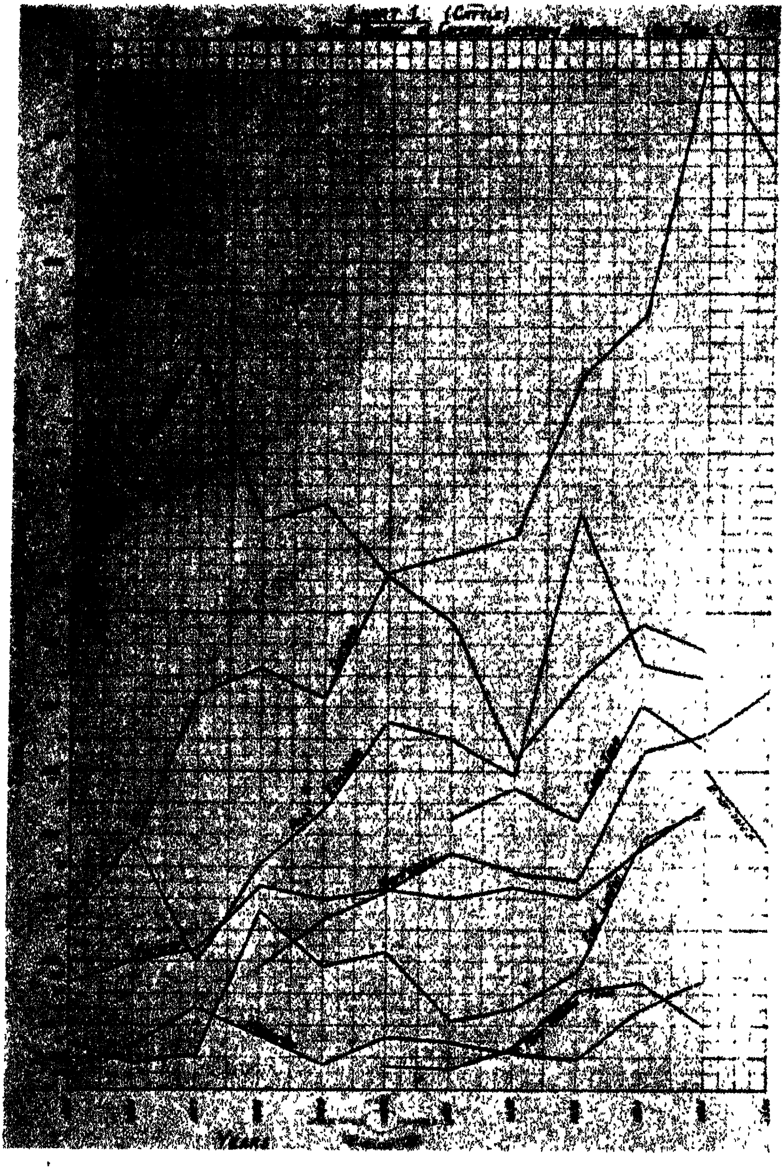

Chart 1 (Cattle).

Photograph of Graph in Dr. H. H. Curson's Note "Measles in Cattle and Pigs". 1936.

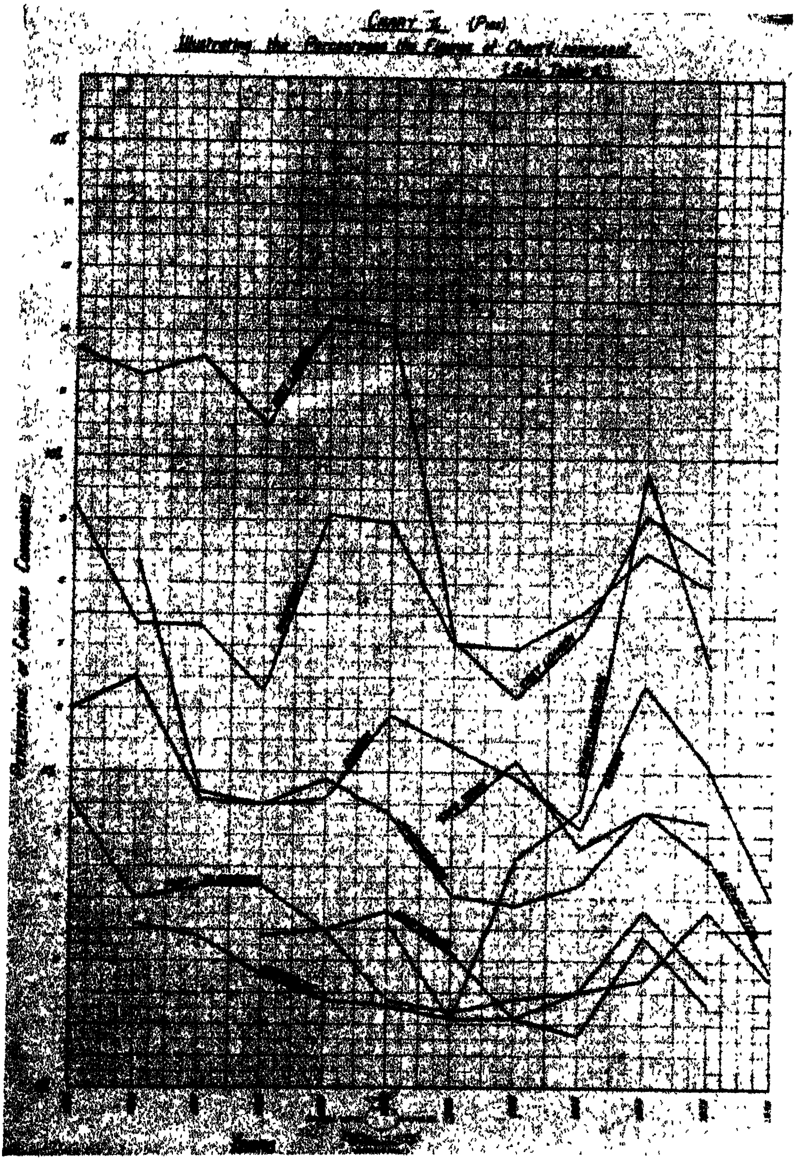

CHART II (PIGS).

CHART II (CATTLE).

Summary.

Reference to the accompanying "Incidence Maps" will clearly show the areas in which the largest percentages of infection are found. These are marked in black, in respect of Pig Measles. The largest Union centres, e.g. Johannesburg, Capetown, Durban, Pretoria, Bloemfontein, Port Elizabeth, East London, Pietermaritzburg, Kimberley and all the Witwatersrand towns obtain their slaughter stock from various parts of the Union, so that it is difficult to fathom the incidence of infection and its origin. A more correct reflection is probably shown by the recordings of the smaller abattoirs, where much of the stock slaughtered is reared locally.

Heavy infection in pigs is reflected in such centres as Ficksburg, Clocolan, Senekal, Wepener and Bethlehem, which are situated close to the Basutoland border, and in the Transvaal a very interesting "black zone" may be traced from the North-West Cape (Mafeking and Vryburg), through Lichtenburg, Potchefstroom, Rustenburg, Pretoria, Witbank to Middelburg. It is correct to state that a large percentage of pigs slaughtered in this "black zone" originates from native areas. The Vryburg and Mafeking Districts have numerous Native Reserves; Lichtenburg District has a large Reserve near Delarey, many native-owned or leased farms, and the district adjoins the vast Moiloa Native Reserve of Marico; Potchefstroom has numerous native farms; Rustenburg District, likewise, has many Native Reserves; Pretoria and Witbank Districts have many native areas, and Sekoekoeniland forms a considerable portion of Middelburg District. It is with regret that no figures are available for the Transkeian Territories, but relatively high percentages were obtained from Kingwilliamstown, close by.

The incidence of *C. bovis* is highest in Natal, the extreme Eastern Transvaal and also in the Eastern Cape, namely at the abattoirs at Kingwilliamstown, East London, Port Elizabeth and Fort Beaufort, and it can safely be presumed that a large percentage of the bovines slaughtered at those abattoirs are of native origin.

The following tables show a summary of the average percentages, in reverse order of frequency, of the incidence of *C. cellulosae* and *C. bovis* at South African abattoirs. The averages given are over periods ranging between 1 and 10 years.

Average Percentages of C. cellulosae *at Union abattoirs.*

Aliwal North	0·5	Dundee	2·70
Beaufort West	0·67	Bedford	2·75
Heilbron	0·88	Middelburg (C.)	2·9
Germiston	1·04	Vryheid	2·9
Mossel Bay	1·37	Swellendam	3·0
Stellenbosch	1·58	Uitenhage	3·0
Kimberley	1·68	Queenstown	3·3
Greytown	1·69	Paarl	3·33
Pietermaritzburg	1·77	Graaff-Reinet	3·33
Port Elizabeth	1·77	Springs	3·91
Worcester	1·97	Randfontein	3·96
Ladysmith	2·0	Capetown	4·26
Bloemfontein	2·13	Kroonstad	4·26
Newcastle	2·45	Brakpan	4·27
Cradock	2·49	Malmesbury	4·27
George	2·61	Winburg	4·4

Johannesburg	4·42
Harrismith	4·51
Kingwilliamstown	4·70
Klerksdorp	4·91
Pietersburg	4·95
Volksrust	5·0
Nigel	5·02
Frankfort	5·03
Durban	*5·16
Parys	5·51
Riversdale	5·8
Lindley	6·0
Krugersdorp	6·10
Nelspruit	6·41
Mafeking	6·67
Vryburg	7·0
East London	7·67
Pretoria	7·85
Witbank	8·97
Clocolan	9·0
Fort Beaufort	9·29
Wepener	9·43
Rustenburg	10·12
Middelburg (Tvl.)	11·49
Bethlehem	15·2
Potchefstroom	15·3
Lichtenburg	19·48
Ficksburg	25·0
Senekal	25·07

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* Durban.—The percentages given for Durban, of *C. cellulosae* and *C. bovis* in the accompanying maps, viz., 3·07 and 4·16 respectively, were for the 12 months ended June, 1936. At the time the maps were compiled, I did not have all the data for Durban. The percentages shown in the tables, viz., 5·16 in pigs and 2·68 in bovines, are the average percentages for 10 years ended 1936.

Average Percentages of C. bovis *at Union abattoirs.*

Heilbron	0·08
Newcastle	0·158
Lichtenburg	0·18
Beaufort West	0·25
Vryburg	0·3
Worcester	0·31
Malmesbury	0·32
Kroonstad	0·45
George	0·53
Uitenhage	0·6
Queenstown	0·67
Brakpan	0·71
Johannesburg	0·75
Cradock	0·75
Burghersdorp	0·87
Upington	0·89
Middelburg (C.)	0·89
Ficksburg	1·09
Volksrust	1·06
Capetown	1·12
Springs	1·20
Kimberley	1·22
Boksburg	1·22
Potchefstroom	1·23
Klerksdorp	1·37
Krugersdorp	1·46
Germiston	1·48
Aliwal North	1·5
Bedford	1·5
Parys	1·51
Paarl	1·75
Pretoria	1·98
Clocolan	2·0
Senekal	2·01
Lindley	2·10
Nelspruit	2·03
Frankfort	2·03
Bethlehem	2·13
Randfontein	2·30
Stellenbosch	2·44
Mafeking	2·67
Durban	*2·68
Witbank	2·75
Nigel	2·80
Pietersburg	2·97
Riversdale	3·0
Middelburg (Tvl.)	3·04
Greytown	3·84
Ladysmith	4·0
Swellendam	4·0
Vryheid	4·3
Bloemfontein	4·87
Brandfort	5·0
Rustenburg	5·18
Kingwilliamstown	5·2
Pietermaritzburg	5·3
Barberton	5·31
East London	5·69
Dundee	5·8
Fort Beaufort	6·1
Port Elizabeth	7·29

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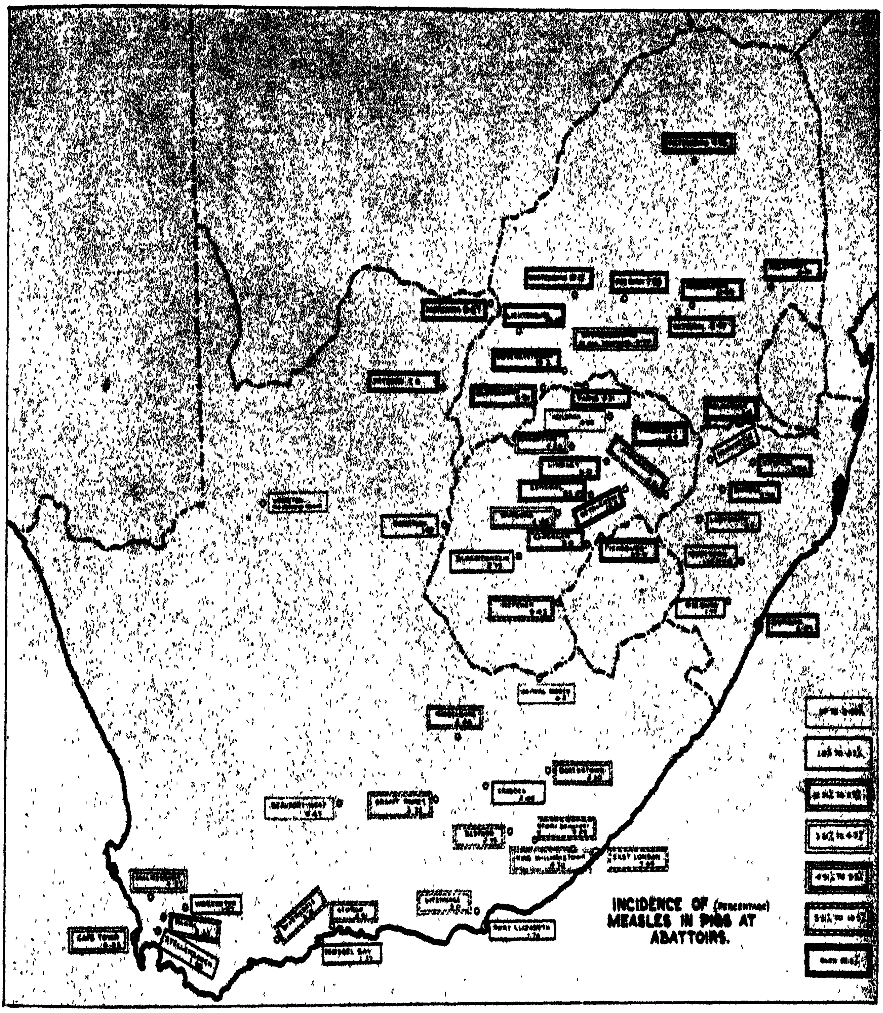
* Durban.—The percentages given for Durban, of *C. cellulosae* and *C. bovis* in the accompanying maps, viz., 3·07 and 4·16 respectively, were for the 12 months ended June, 1936. At the time the maps were compiled, I did not have all the data for Durban. The percentages shown in the tables, viz., 5·16 in pigs and 2·68 in bovines, are the average percentages for 10 years ended 1936.



Map I.

Showing average percentages *C. cellulosae* at Union Abattoirs.

MAP II.

Showing average percentages *C. bovis* at Union Abattoirs.

PART III.

In his "Text-book of Meat Inspection" von Ostertag divides the animal parasites found in meat inspection into (i) those not transmissible to man through the consumption of meat, and, (ii) those parasites transmissible to man through the consumption of meat. In the latter category he mentions but three parasites:

(*a*) *Cysticercus inermis (bovis)* in cattle.

(*b*) *Cysticercus cellulosae* in swine.

(*c*) *Trichinella (trichina) spiralis* in swine.

An endeavour will be made in Part V of this article to show the import of the two first-named parasites on hygiene, with special reference to South African conditions.

The *Trichinella spiralis* will receive no further consideration in this work, since it is neither a *cysticercus*, nor has its occurrence in South Africa been recorded.

On the other hand, there is a number of *cysticerci* found in the viscera and tissues of slaughter animals, including pigs and bovines, which, although meat may be condemned owing to their presence, will not give rise to an adult tapeworm in man. Some of these *cysticerci*, however, are of importance when arriving at a differential diagnosis, and will be mentioned in that section.

A. Morphology, Development and Life Cycle.

1. CYSTICERCUS CELLULOSAE.

The *Cysticercus cellulosae* is a round to oval bladder, which in young specimens may frequently be spherical, but in older specimens it generally has an oval or even elliptical shape. The maximum recorded size is 20 mm. by 10 mm.

It consists of (*a*) the so-called bladderworm capsule, or caudal vesicle, (*b*) the parasitic head and neck, or *scolex*. The actual bladderworm is enveloped in a whitish-gray transparent outer connective tissue membrane, formed by the host tissues as a protective reactor against the surrounding muscular tissue of the host. The caudal vesicle consists merely of an outer cuticle and a subcuticular layer. The caudal vesicle is easily removed from the connective tissue capsule, and is of delicate structure, containing a variable amount of fluid. It is very transparent, showing the invaginated *scolex* inside the bladder. One portion of its wall is narrowly folded to form, what was formerly called, and is still so-called by some writers, the *receptaculum scolicis*. The *scolex* is invaginated into the *receptaculum*, being attached to the fold of the vesicle by its neck portion. The opening or "*hilus*", into which the *scolex* is invaginated, is extremely narrow and barely visible. The *scolex* and its *receptaculum*, in the early stages of development grow in unison, but after a time the *scolex* outgrows its *receptaculum*.

The resultant restriction to the longitudinal growth of the *scolex* causes it to form a bow or bend into an S-, or 6-shape, as Yoshino (1934) describes it. This constriction causes the comparatively long neck of the *scolex* to make one-and-a-half complete coils, and transversely folds it in the invagination process to resemble a closed concertina. The neck is transversely striated and contains numerous calcareous corpuscles, which are characteristic of tapeworm tissue. Ransom suggests that the calcareous corpuscles are composed of calcium albuminate. When treated with acid, however, the corpuscles dissolve, giving off gas, and they may, therefore, probably be composed of calcium carbonate.

The size of the head varies with the maturity of the *Cysticercus*. When mature it measures from about 0·6 mm. to 1mm. in diameter. The head is spherical in shape. Its dome is formed by a prominence, the *rostellum*, a strongly muscular structure, which is capable of contraction and expansion. The *rostellum* measures about 0·36 mm. in height and diameter, and is armed with two rows of hooks. Owing to its contractive and expansive powers the *rostellum* may sometimes be deeply sunk into the wall of the head, and sometimes protruded into a dome-like proboscis. Four prominent suckers are situated antero-laterally, with the *rostellum* approximately equidistant from each. They measure from 0·4 mm. to 0·5 mm. in diameter. Leuckart, in describing the range of movement of the suckers, mentions that " the whole four may be raised up like arms, extend in different directions, and then contract. This is very marked when the suckers feel about in front as though trying to fix themselves to some object situated in front of the head. As often as this motion takes place the apex is observed to sink in, and to remain in this position until it again protrudes and allows the hook-apparatus to unfold itself ".

FIGURE 1.

C. cellulosae head, showing rostellum with hooks, and on the left two suckers prominently, other two suckers less prominent. This scolex evaginated artificially after 28 days cooling.

Photograph by Director of Veterinary Services, Onderstepoort. Magnfn. 40×.

Specimen, Bloemfontein Municipal Abattoir.

FIGURE 2.

Completely evaginated scolex of *Cysticercus* mentioned under Figure 1.

Magnfn. 7×.

The double row of hooks is arranged into a circle on the *rostellum*. The anterior hooks are the larger, and the posterior, or smaller hooks always individually occupy the spaces between each anterior pair of hooks, thus alternately there are large and small hooks. The shape of the hooks is characteristic and a distinct " handle ", " guard " and " blade " can be recognized. The root processes are thick and the tips curved. The total number of hooks in *Cysticercus cellulosae* is from 22 to 32. Yoshino found the number of hooks in the specimens examined by him to be 22, 24, 26 or 28. The large hooks measure from 0·16 mm. to 0·18 mm. and the small hooks 0·11 mm. to 0·14 mm. (Yoshino's measurements are 0·128 mm. to 0·162 mm., and 0·100 mm. to 0·125 mm., respectively.)

In describing the development of the hooks on the *rostellum*, Yoshino states that they differ in shape according to the development of the *Cysticercus cellulosae*: " The initial hooks on the rudiment of the *rostellum* in *C. cellulosae* about 40 days old are like needles or spines, and gradually they curve outwards like the horns of cattle ".

The histological structure of the *scolex* of *Cysticercus cellulosae* about 40 to 50 days old is similar to that of the mature *Cysticercus*, and there are recognizable two pairs of excretory canals and very many calcareous corpuscles in the muscular layer developed from the spindle cells in the first cell layer.

In the adult *Taenia solium*, the head is joined to the body or *strobila* by a neck, usually four times as long as the head itself. The *strobila* is segmented, the segments being called *proglottides*, which, in the case of the mature segments, are longer than broad. The *proglottides* are formed behind the neck, in a continuous chain. The anterior segments are the youngest, and push the older, mature and gravid *proglottides* farther and farther to the posterior of the host's intestine. The total length of the worm is about 3·5 m. and in rare cases up to 8 m.

The structure of the *strobila* is simple. Externally it possesses a cuticular layer, which contains numerous fine pores, through which the parasite absorbs its nourishment. Immediately below the cuticle is a subcuticular layer, with a layer of cells on its inner surface. Below the cuticle are layers of delicate transverse and longitudinal muscles. The interior of the body consists of the parenchyma, which is divided by a strong layer of transverse muscles into cortical and medullary portions. The cortical portion of the parenchyma contains numerous oval calcareous corpuscles, similar to those mentioned in the description of the *scolex*, and are distributed throughout the length of the *Taenia*. They may measure up to 0·019 mm. In the medullary portion are situated the excretory, nervous and reproductive systems. All tapeworms are devoid of an alimentary canal, absorption of nourishment taking place through the pores in the cuticle, and excretion through " flame-cells " and a pair of dorsal and ventral longitudinal canals on either side of the *strobila*. At the posterior part of each *proglottis*, each pair of canals is joined by a transverse canal, and in the *scolex* itself all canals are joined by transverse loops.

The nervous system consists of two large longitudinal and several smaller nerve trunks, which run throughout the *strobila*. These are joined by several ganglia and transverse commissures at the *scolex*.

The *Taenia solium* and all *taeniae* are hermaphrodite parasites. The male reproductive organs are the first to appear in young *proglottides*. They consist of: (i) Numerous *testes*, which secrete into (ii) *vasa efferentia* uniting to form a (iii) *vas deferens*, which forms a (iv) *seminal vesicle*, and ends in (v) a *cirrus-pouch*, containing a *cirrus*, opening at the genital pore, which lies in a sinus on the lateral margin of the *proglottis*, close to the genital pore of the female organs. The genital pores are situated on irregularly alternate margins in successive *proglottides*.

The female generative organs consist of: (i) A *vagina*, opening at the genital pore. It is a narrow tube, bearing (ii) a *seminal vesicle*, and ends in the (iii) *ootype*, surrounded by Mehlis' gland, where it is joined by the oviduct and the vitelline duct. (iv) The *ovary*, which is single and lobed, is situated at the posterior part of the *proglottis*. (v) The *ootype* is also in contact with the *vitelline gland*. (vi) The *uterus* is characteristic, and one of the main differential diagnostic features of the species. It has a median longitudinal stem with 7 to 12 lateral branches.

Self-fertilization may take place in each individual sexually mature *proglottis*. Tapeworms may also bend double and the male products developed earlier in younger *proglottides* may fertilize the female products developed later in older *proglottides*.

The embryonic egg-shell is thick and radially striated, spherical, rarely ovoid in shape, and measures 0·042 mm. in diameter. Leuckart's measurements were 0·06 mm. and those of Yoshino 0·043 to 0·068 mm. The embryonic development takes place in the uterus, and the eggs contain a spherical embryo, with three pairs of hooks, the *hexacanth embryo*. The embryo with its embryonic egg shell is known as the *oncosphere*. Yoshino (1934) found that under abnormal circumstances the embryo may have 8, 10, 12, 14, 16 or 18 hooks, instead of 6.

Gravid proglottides measure 10 to 12 mm. long by 5 to 6 mm. broad. The gravid segments are passed to the exterior by the human host, in his excretum, and are frequently detached in short chains. The worm may live in man for years, and sometimes more than one individual may be present in one host. Yoshino (1934) intentionally infected himself with *Taenia solium* and studied many gravid *proglottides*. He found that the gravid proglottides just discharged move about, alternately extending their anterior ends then contracting themselves. "During the extensions the eggs are pushed out from the uterus through the anterior end of the *proglottis*. With the extensions and contractions from 31,000 to 55,300 eggs are evacuated and only 480 to 1,500 eggs remain in the *proglottis*," he wrote. Leuckart found that the contents of the uterus of each segment was 6 cubic mm., and it held some 53,000 eggs.

Development of Cysticercus cellulosae.

The egg hatches after it has been swallowed by the pig (also by man, the dog, or in at least one instance the monkey, as was reported by Walker), and the *hexacanth embryo* penetrates into the intestinal wall. The migratory course of the embryo in the body of its intermediate host, the pig, was carefully studied by Yoshino in 1933. This writer experimented upon a number of pigs, which he fed with large numbers of eggs of *Taenia solium*. He obtained the following results:—

(1) The eggs of *Taenia solium* hatch in the upper part of the small intestine of the pig and the greater number of freed embryos enters the mucous membrane of that portion, while a smaller number penetrates into the middle or lower parts of the intestines.

(2) The number of hours required for the hatching of the eggs varies, and Yoshino saw freed embryos in the mucous membranes of the intestines 15 to 48 hours after the experimental feeding, and they were also found in the internal organs and muscles within 24 to 72 hours after feeding.

(3) Embryos found in the intestinal wall usually had no hooklets, having lost them by penetrating through the tissues. During the early stages, 15 to 48 hours after feeding, specimens with one or two hooklets could be seen.

(4) The youngest *Cysticercus cellulosae* or transitional forms found in the intestinal wall were spherical or ovoidal in shape. They consist of round cells, and under the microscope appeared grayish-white in colour, and measured 0·024 mm. to 0·03 mm. in length and 0·021 mm. to 0·026 mm. wide. The transitional forms were mostly found in the tunica propria and rarely in the tela submucosa and the muscular layers. In those cases the blood vessels in the tunica propria, into which the transitional forms were about to penetrate, or had penetrated, were congested and enlarged. The youngest *Cysticerci cellulosae* were rarely found in the abdominal cavity of a pig between 24 and 72 hours after the experimental feeding. Those *Cysticerci cellulosae* found in the internal organs or muscles of a pig between 24 hours and 72 hours after experimental feeding were light greenish in colour and spherical or ovoidal in shape. They consisted of round cells. Between 24 and 48 hours after experimental feeding, the size of the *Cysticercus* was 0·024 to 0·042 mm. in length and 0·021 to 0·036 mm. wide. Between 48 and 72 hours after feeding the dimensions were 0·03 mm. to 0·058 mm. long and 0·027 mm. to 0·054 mm. wide.

(5) Between 6 days and 15 hours and 12 days and 15 hours after experimental feeding, the young *Cysticerci cellulosae* appeared macroscopically as almost transparent and colourless spots, and were difficult to find within the muscles and organs, unless they were detached from them. Under the microscope they were light greenish in colour, and spherical, ovoidal or cylindrical in shape. At 6 days and 15 hours after experimental feeding they were generally solid, but larger specimens were somewhat cystic. Those examined 12 days and 15 hours after experimental feeding were cystic, contained

a sticky fluid, " and through the cyst wall a spot could be seen, which might have been the rudiment of the head and its *rostellum* ". (It is extremely unlikely that the last surmisal of Yoshino is correct. In such young stages there would be nothing more than a slight thickening of the wall where the invagination will later occur. The scolex is formed much later at the bottom of the invagination or receptaculum).

(6) At 12 days and 15 hours after experimental feeding the young *Cysticerci cellulosae* were quite large and cystic, and contained fluid. The cyst wall became thinner than in younger specimens and consisted of a cuticular membrane and a subcuticular layer. At a point destined to become the receptaculum, a great number of ovoidal cells began to accumulate, and the cuticular layer of that portion became thicker and curved into the accumulated cell-layers.

At 12 days and 15 hours after experimental feeding the young *Cysticerci* were found in the liver, especially in bleeding areas in the parenchyma. They were also found in the brain, especially in the cortical substance of the cerebrum. Within 12 days and 15 hours after experimental feeding the young *Cysticerci cellulosae* were found in body muscles and heart muscles and were accompanied by round cell infiltrations.

From the above results Yoshino concludes that the embryos hatch in the small intestine of the pig, penetrate into the intestinal wall, and the majority enter the blood stream by the capillaries in that region. They are then carried to the internal organs and muscles, where they develop into *Cysticerci cellulosae*. Others penetrate through the intestinal wall into the abdominal cavity and die there.

On the later stages of development of the *Cysticercus cellulosae* various observers have recorded as follows:—

Hutyra and Marek.

At 20 days *Cysticercus cellulosae* is about the size of a pin head, and the head is visible as a small white point.

At 40 days it appears as big as a mustard seed, and the head may be plainly seen, but it has neither suckers nor hooks.

At 60 days the cyst is as big as a pea, with head with suckers and hooks, but no neck.

After 3 months the " bladderworm " is fully developed and behind the head the transversely striped neck may be seen.

Braun-Seifert.

The complete development of the *Cysticercus cellulosae* takes from 2½ to 4 months.

Mönnig.

The *Cysticercus* requires about ten weeks for its complete development in the pig. After about two months the bladderworm is already infective as the suckers and ~~hooks are~~ sufficiently well developed to allow the scolex to attach itself.

Yoshino.

At 20 to 30 days the *Cysticercus cellulosae* measured 1·1 mm. to 4·1 mm. by 0·8 mm. to 3·2 mm.

At 40 to 50 days dimensions were 3·4 mm. to 8·2 mm. by 2·9 mm. to 6·0 mm.

At 60 to 70 days dimensions were 5·6 mm. to 8·5 mm. by 3·1 mm. to 6·5 mm. At the last named stage it might be fully developed and infective, but may still increase in size and measure 8·0 mm. to 14·5 mm. in length, by 4·5 mm. to 8·0 mm. in width between 254 and 325 days after feeding.

Twenty days after experimental feeding the rudiment of the scolex may become gradually distinguishable.

Forty to fifty days after feeding the scolex has developed fully with four suckers arising from its invaginated surface, and in its blind end the rostellum provided with hooks is formed. It measures from 0·83 mm. to 1·97 mm. in length.

In *Cysticercus cellulosae* 60 to 70 days old, the scolex is fully developed and its elongated neck is bent within its receptaculum, showing on its invaginated surface many foldlike septa, because the scolex grows much more rapidly than the receptaculum. According to the development of the bladder the neck of the scolex elongates rapidly, and on its invaginated surface numerous fold-like septa appear. Outgrowing the receptaculum, it bends as a whole into an S-, or 6-like shape and the receptaculum becomes a thin membrane.

The histological development of the head, according to Yoshino, is as follows:—

At 20 days the rudimentary scolex is a simple tube, consisting of cuticle and subcuticular layer. The subcuticular layer may again be divided into an outer (first cell) layer, which directly joins the cuticle, and consists of spindle cells, and an inner layer (second cell layer) of polymorphic cells. At 40 to 50 days the scolex is almost fully developed and is provided with four suckers and the rostellum. In this stage the suckers are hemispherical and measure 0·225 mm. to 0·352 mm. in diameter. Still later, at 60 to 70 days, the suckers measure 0·325 mm. to 0·384 mm. in diameter.

II. CYSTICERCUS BOVIS.

The general structure of the *Cysticercus bovis* and its resultant adult *Taenia saginata* resembles the *Cysticercus cellulosae-Taenia solium* closely, with the following enumerated points of difference:—

(*a*) Intermediate host, the ox; very rarely man. Adult host, man only.

(*b*) The outer connective tissue membrane is very much thicker than that of *Cysticercus cellulosae*, and much more firmly attached to the caudal vesicle.

(*c*) The bladder itself is much less transparent, and contains a thicker fluid, which is frequently more turbid than in *Cysticercus cellulosae*. The scolex is, therefore, less visible.

(*d*) The bladder is decidedly more greyish in colour, and very frequently the fluid contents give the bladder a reddish-brown tint. Piettre (1922) gives the opinion that the red colouration may be ascribed to the absorption of haemoglobin from the surrounding muscles. Valade (1927) suggests that the reddish tint apparently results from histolysis of muscle fibres as the result of the excretion of toxic materials by the scolex.

(*e*) The *Cysticercus bovis* usually measures 7·5 mm. to 9 mm. by 5·5 mm. when fully developed.

(*f*) The scolex, especially in the adult stage is very much larger than that of *Cysticercus cellulosae-Taenia solium*. It is 1·5 mm. to 2 mm. in diameter.

(*g*) The embryo is a hexacanth (six-hooked) larva, but neither in the *Cysticercus* nor in the adult stage has the scolex a rostellum and hooks.

(*h*) The four suckers are even more muscular than those of *Cysticercus cellulosae-Taenia solium*, are larger, with unusually thick walls. As compensation for the absence of hooks, the suckers are capable of greater suctorial attachment.

(*i*) Pigmentation around the suckers is very well-marked in the adult *Taenia saginata*, and gives the worm the appearance of possessing a big black head. Pigmentation occurs to a much less extent in *Taenia solium*.

(*j*) The adult tapeworm is much longer than *Taenia solium*, and may measure from 4 to 10 m. in length.

(*k*) Gravid proglottides are from 16 to 20 mm. in length and from 4 to 7 mm. wide.

(*l*) Gravid segments are generally voided singly, very rarely in chains, and may sometimes be liberated spontaneously to the great discomfort and embarrassment of the human carrier.

(*m*) The gravid uterus has 15 to 35 lateral branches on either side.

(*n*) The embryonic egg-shell is ovoidal, rarely spherical, and measures 0·045 mm. by 0·043 mm.

(*o*) Malformations are quite common in *T. saginata*. These may take the form of specimens with multiplication of the generative openings. (Leuckart.) Supernumerary joints, and sometimes duplication of strobilae have been recorded. Palais (1933) described a specimen obtained from Brazil, in which the two strobilae were attached one at right angles to the other. Leuckart states that he found only one case of malformation in *Taenia solium*.

(*p*) The worm has been known to live in man for 20 years. Very rarely is more than one specimen found in one host, and in that respect Leuckart was correct when he took exception to the name *Taenia solium* (solitary tapeworm), and pointed out that *Taenia saginata* was by far the more solitary.

(*q*) With regard to the development of *Cysticercus bovis*, Braun (1900), quoting Hertwig, gives the following table:—

Age of cysticerci in weeks.	Connective tissue		Cysticerci.		Scolex./mm.	
	Length /mm.	Breadth /mm.	Length /mm.	Breadth /mm.	Natural.	Extended artificially.
4	4·0	3·5	2·25	2·25	0·5	0·7
6	4·2	3·5	3·0	2·5	1·0	1·3
8	4·5	3·5	3·25	2·75	1·5 × 1·0	2·9
10	5·0	3·75–4·0	3·5	3·5	1·7 × 1	3·3
12	5–6	3·75–4·0	4·0	4·0	1·8 × 1·0	3·5
14	6·0	4·5	5·0	4·5	2 × 1	4·0
16	6·0	4·5	5·0	4·5	2 × 1	4·25
18	6·25 × 7·00	4·5	6·0	4·5	2 × 1·25	5·0
22	6·5–8·0	4·5	6·0	4·5	2·25 × 1·75	5·5–6·25
28	7·5–9·0	5·5	7·0	5·0	2·5 × 2	7·0

(*r*) *Cysticerci* take about 18 weeks to attain full development. It is usually taken that a diagnosis of cysticercosis will be made in meat inspection from the 6th week onwards—both in the case of *C. bovis* and *C. cellulosae*.

SECOND, OR ADULT STAGE OF LIFE-CYCLE OF BOTH SPECIES.

If man eats viable measly pork or beef, which may be undercooked, or insufficiently cured in the case of ham, the adult stages of the respective parasites are commenced within him.

The bladderworm is swallowed, and within 24 hours, as a rule, the scolex evaginates from the surrounding caudal vesicle into which it had been invaginated. The evagination is caused by the stimulation of the head by digestive juices which permeate through the "hilus" of the invagination. The head attaches itself to the mucosa of the intestine, by means of its hooks and suckers (*Taenia solium*), or suckers only. (*Taenia saginata*).

After having obtained lodgment by means of the scolex, the tapeworm grows, and from the neck the strobila develops. The mature and gravid segments are pushed further to the posterior by the younger segments. Self-fertilization may occur within the proglottides, or proglottides may fertilize one another, and gravid segments are voided.

Contrary to the opinions in many text-books, Yoshino (1934), who examined stools for *Taenia solium*, and Alcaraz (1932), Pardina (1932) and Franzani (1933), who examined stools for *Taenia saginata*, found that in the majority of cases numerous eggs were found in the faeces, whereas comparatively few remained in the voided proglottides. Pardina explains the fact that the detached gravid segments extrude eggs through a ruptured uterine branch. Alcaraz, referring to *Taenia saginata*, explains that segments are expelled singly, causing rupture of the uterus, followed by active expulsion of the ova. Kouri and Basnuevo (1933) found that eggs of *Taenia saginata* were observable in 80 per cent. of stool examinations, in infected cases.

Moore (1916) observed the rate of growth of a *Taenia saginata* in a student at the Potchefstroom (Transvaal) School of Agriculture. He gave the student a vermifuge, which caused the patient to excrete a length of tapeworm, which, according to its appearance, gave Moore the opinion that the entire worm minus its head and neck had been passed. Some time later, the patient was again troubled with the tapeworm, and a second vermifuge was administered, with the result that the entire worm was passed. The time between the first and second vermifuges was 72 days, and the length of worm passed on the second occasion was 19 ft, 3 in. Moore thus estimated that all but the head and neck grew in that time.

When the pig, or the ox, ingests the eggs of the respective species of which it is the intermediate host, the life-cycle is resumed.

B. The Hosts and Pathogenicity of Cysticercus cellulosae.

In the adult stage the *Taenia solium* has only been known to develop in man. Young, immature *Taenia solium* may, however, live for a very short period in dogs and possibly in some other carnivores.

Experimentally we tried to infect six dogs, a jackal (*Thos mesomelas*) and a baboon with *Taenia solium* at the Bloemfontein Municipal Abattoir. In none of these subjects did ripe proglottides or ova pass in the faeces. Dogs Numbers 1 to 4 were destroyed and examined 90 days, 70 days, 60 days and 30 days, respectively, after having been fed large numbers of *C. cellulosae*. Dog Number 5 was killed 3 days after feeding, and dog Number 6 was killed 24 hours after feeding. In not a single case were mature, or immature *Taeniae solium*, or even evaginated scolices observed.

Similarly, all attempts to infect the jackal failed. At various periods, over four months, he received countless thousands of viable *Cysticerci cellulosae*. Since infection over a prolonged period did not result, he was finally given some pork containing numerous viable *Cysticerci cellulosae,* and destroyed two days later. Post-mortem examination revealed several mature *Taeniae marginata* (*hydatigena*), and many thousands of *Echinococci granulosus,* but not a trace of *Taenia solium.* The infections with *T. marginata* and *E. granulosus* had resulted from experimental feedings with numerous *Cysticerci tenuicollis* and *Echinococcus* cysts, which had been administered when we first obtained the jackal, about four months previously.

Infection tests were contemporarily tried on an adult male South African baboon. The baboon was kindly presented for experimental purposes by the Chairman of the Parks Committee and the Curator of the Bloemfontein Municipal Zoological Gardens. He was well housed at the Abattoir, and his diet consisted mainly of fruit, vegetables and bread. He refused to eat meat, whether raw or cooked. At first he was given about fifty *Cysticerci cellulosae* hidden in bread, but, with the natural wiliness of his kind, he frequently broke the bread into crumbs and removed all traces of *cysticerci.* Measles were then stuffed into the pulp of bananas by means of a sharp stick or a pencil, and the canal thus formed was again closed over, so that the baboon could neither detect the presence of the measles in the bananas, nor could he notice that the bananas had been interfered with. He took the bananas readily and by this means approximately 750 viable *Cysticerci cellulosae* and a few *C. bovis* were fed to him, over a period of four months. In order to ensure that only live measles were fed to him, we always tested viability of the *cysticerci* from the same pigs in 5 per cent. sodium taurocholate solution and by actual infection tests on a human subject, according to Keller's and Iwanizky's methods. At no time did our baboon excrete *Taenia* segments. Four months after the original feeding the baboon died from acute pneumonia, contracted during a sudden cold and wet spell. A careful post-mortem examination was made, which revealed pneumonia, but not a single tapeworm, mature or immature, was found.

Under natural conditions, the baboon is not carnivorous in the true sense of the word, although he may feed on locusts, scorpions and grubs. In a few instances they have been known to attack flocks of sheep, causing wilful destruction. A favourite practice of these marauding troops of baboons is to disembowel sheep, and leave the carcasses on the veld, but it is very doubtful if they will at any time attack and make a meal of pigs. It is, therefore, most unlikely that the baboon will acquire natural infection of *Taenia solium.* Although our experiments were numerous attempts to infect one baboon, it can reasonably be concluded that the baboon is immune to infection with *T. solium,* even with attempts at artificial infection. It is also very unlikely that any of the higher anthropoid apes are subject to the parasite. In conclusion, it may be mentioned that in 1932 Clarenburg recorded that he failed to infect various monkeys with *Taenia saginata.* His subjects were fed several *C. bovis,* fresh specimens, as well as some which had been preserved in a cooler for three weeks.

In the cystic stage a number of animals has been named as intermediate hosts. Authentically it is accepted that the pig, man, the dog and recently, the monkey are definite hosts. In addition it has been mentioned by some writers that *C. cellulosae* was found in sheep, goats, cattle, horses, antelopes, deer and bears, " but the identification of the *cysticerci* was undoubtedly erroneous in many cases ". (Mönnig, 1934.)

The *cysticerci* found in sheep and goats were very probably *C. ovis* which closely resembles *C. cellulosae*, and has a rostellum bearing 24-36 hooklets. Von Ostertag (1934) mentions that Ciurea examined seven cases of suspected *C. cellulosae* in sheep and found that they were actually typical cases of *C. ovis*.

In cattle and wild buck they may have been the *Cysticercus* of *Taenia hyaenae*, a tapeworm from the hyena. In 1932 Martinaglia encountered a peculiar measle in a bovine carcass at the Johannesburg Abattoir. " The *cysticerci* were armed and unlike the bovine bladderworm. On further identification Dr Mönnig of the Veterinary Research Laboratory, Onderstepoort considered the hooks of this *cysticercus* resembled those of *T. hyaenae* ".

It is possible that the *cysticerci* found in equines, antelopes, etc., were mistaken by some writers for *C. cellulosae*.

Some years ago, during three years' service in the wilder parts of the Bechuanaland Protectorate, the present writer found what he took to be *C. cellulosae* in two African bush pigs (*Potamochoerus choeropotamus*). Both wild pigs were shot by native attendants, and in curiosity the writer inspected the carcasses, which were found to be heavily infested with measles, which closely resembled those of the domestic pig. Unfortunately, owing to his remoteness from civilization at the time, the writer was unable to examine the *cysticerci* microscopically, or, since it was also impossible to send specimens away, owing to no preservatives being available, it was impossible to have them definitely identified. Dr. Mönnig mentioned to the writer that Mr. Harris, who was engaged on the Government's tsetse-fly campaign in Zululand, reported similar cases to him. According to Daubney (1936), up to date there is no record of *C. cellulosae* from any of the wild pigs of East Africa.

INFECTION IN THE PIG.

The infestation of the pig with *Cysticercus cellulosae* is usually of a very heavy and generalized nature. In this respect it often differs from the infestation of the ox with *C. bovis*.

In the pig " predilection sites " are sometimes mentioned, but that term, in South Africa, is really only applicable in the exceptional cases of light infestation.

At the Bloemfontein Abattoir we made a systematic study of so-called " predilection sites " in order to ascertain whether these sites corresponded with those described by older overseas authors.

During the calendar years 1935 and 1936, 180 pigs were found measly. Of this number the great bulk were grossly infested, and only 30 had less than 10 measles in the routine inspection incisions. The ratio of 5 : 1 heavily to lightly infested carcasses was more or less fairly representative of infection in other parts of South Africa. It is interesting to record that from Swellendam an exception to the rule was reported. The Abattoir Superintendent of that small centre advised me that he had noticed considerably more pigs lightly infested in the Swellendam abattoir than during his previous service in abattoirs in the Transvaal and Northern Orange Free State.

In the subjoined table the ratios of heavily infested to lightly infested carcasses are given for some centres in the Union. The ratios given are only in respect of such centres where actual observations were recorded and the details were available. A number of superintendents of other abattoirs, who did not keep actual records, advised me that in general the nature of infestation was very heavy, and lightly infested cases were rare.

Table showing ratio of heavy infestation to light infestation.

Ladysmith (Ntl.)....	3 : 1	Rustenburg......	100 : 0·2	Graaff-Reinet.......	12 : 1
Newcastle...........	7 : 4	Bloemfontein.......	5 : 1	Middelburg (C.)....	4 : 1
Potchefstroom.......	7 : 3	Kroonstad.........	3 : 1	Port Elizabeth.....	52 : 1
Germiston..........	3 : 1	Bethlehem.........	19 : 1	Queenstown........	19 : 1
Klerksdorp..........	13 : 2	Fort Beaufort......	8 : 1	Riversdale.........	7 : 1
Middelburg (Tvl.)....	10 : 1	George............	7 : 3	Uitenhage..........	19 : 1
Nigel...............	5 : 1				

The Superintendent of the Fort Beaufort abattoir made the following observation: 1 cyst 1 carcass; 2-5 cysts 2 carcasses; 5-10 cysts 2 carcasses; over 10 cysts 40 carcasses.

Von Ostertag (1913) gives the following table showing the ratio of heavily infested carcasses to lightly infested carcasses at the Berlin Abattoir:—

Year.	Total number of measly pigs.	Extensively infested.	Lightly infested.
1895–96............................	627	304	323
1896–97............................	509	251	258
1899...............................	325	118	207

Judging from the above table, it would appear that in Germany, about 40 years ago, the ratio of lightly infested pig carcasses to heavily infested pig carcasses was found to be slightly higher in favour of light infestation. Von Ostertag, however, points out that the ratio of heavy infestation is much higher in the case of hogs than in the case of bovines with *C. bovis*.

Le Coultre (1928) gives the following ratio obtained during his investigations in the Netherlands East Indies in 1927:—

Boeleleng: 36 cases; 17 heavily infested; 19 lightly infested.

Denpasar: 23 cases; 18 heavily infested; 5 lightly infested.

Makassar (1926): 85 cases, all with less than 13 measles.

Soerabaia: All cases heavily infested.

In the usual cases of infestation with *C. cellulosae*, pigs may harbour many thousands of parasites. It is quite common that hardly a fraction of an inch of the carcass may be noticed free from bladderworms.

In order to arrive at a quantitative estimate of the number of bladderworms in heavily infested pig carcasses, I caused a count to be made in small pieces of meat from two heavily infested pigs at Bloemfontein abattoir. In each case the head was removed, the vertebrae and the bones of the limbs were stripped of meat, and the actual mass of meat itself weighed. The first pig weighed 75 lb. stripped, and the second, a fairly large pig, 150 lb. No viscera of any kind were included in the weights. In each case a small piece of pork, 1 inch cubed, was excised from the deep-seated musculature on the medial aspect of the thigh. Each small piece of meat weighed ¾ ounce. In the first piece we found 80 measles, which represented some 128,000 *cysticerci* in the pig, minus its head, heart, liver, brain and tongue, all sites where many measles might have been found. In the second piece we found 184 measles, which represented some 588,800 *cysticerci* in the 150 lb. pig, minus its head and viscera.

Hall (1920) found 70 *cysticerci* in a small piece of pork which weighed 5 grammes. Thus he estimated that so many thousand measles were to be found in the entire carcase.

Küchenmeister found 133 measles in 17 grammes of pork, representing about 80,000 to the kilogramme or 2¼ lb.

At Bloemfontein we judged all carcasses is which more than 10 *cysticerci* were found, in the routine incisions, as heavily infested. During the calendar years 1935 and 1936, 150 heavily infested pig carcasses were found. Statistics are not available of the predilection sites in all the heavily infested carcasses. More accurate observations were made in respect of the thirty lightly infested carcasses. Ten heavily infested pig carcasses were, however, carefully surveyed. Measles were invariably found scattered throughout the musculature of the carcasses. They were more densely located in the muscles above the elbow and in the neck muscles. Infection of the thigh muscles (leg of pork) was very heavy, and invariably numerous measles were found in the perineal region. Although in the ten heavily infested pigs, closely examined, a fairly heavy infestation of the masticatory muscles was observed, it was relatively less heavy than would have been the case of gross infestation of *C. bovis* in the ox. It will be observed that not a single measle was found in the masseters or the pterygoid muscles of the 30 lightly infested pig carcasses. (See predilection sites in light infestations.)

In three of the ten observations *Cysticerci cellulosae* were found in the fat and subcutaneous tissues. The abdominal muscles, intercostals and cervicals were invariably infested. As a rule the degree of infestation was lighter in the abdominals and intercostals than in the cervicals, where usually, numerous cysts were found. Other sites in which measles were invariably found in the ten observations were, in order of density: the psoas muscles; the sub-vertebral muscles; the tongue; the heart; the oesophageal musculature; the diaphragm. In six out of the ten observations *cysticerci* were found in the brain; in two out of the ten observations in the eyeball and conjunctiva. In none of the special observations were *cysticerci* found in the testicles of boars, or in the vagina and uterus of sows, respectively. (Of the ten special observations, two were boars, five were sows and three were castrates.) On at least one other occasion in each gender did we find measles in the respective generative organs. Intra-uterine infections of foetuses were never observed, although on a few occasions pregnant -sows were inadvertently slaughtered.

In the six cases of cerebral cysticercosis, cerebral *cysticerci* were found singly in 2 cases, six cysts in 1 case, eight cysts in 1 case, and numerous cysts in 2 cases.

Predilection Sites in Light Infestations.

In most countries regulations governing meat inspection lay down standard routine incisions which are to be made into carcasses, with a view to inspection for *cysticerci*. Such routine incisions are made into a carcass so as to expose surfaces where measles are most frequently found, without mutilating the carcass. In lightly infested pig carcasses it is quite possible to miss *cysticerci* which may be present, but have not been exposed during the routine inspection.

Irvine-Smith (1911), in " The Report of the Director of Abattoirs and Live Stock Market, Johannesburg, 1910-11 ", mentions that during that year under report two pig carcasses, bearing the abattoir " passed " stamp, were afterwards found in butcher shops to be measly. In the one case measles were found by the butcher himself, and in the other case by one of the Municipal Health Inspectors. Col. Irvine-Smith points out that it would have been difficult to detect measles in those cases, without mutilating the carcasses.

For the inspection of pig carcasses for *cysticerci* in South Africa, Regulations have been framed under Section 115 of the Public Health Act, No. 36 of 1919 (Government Notice No. 2118 of 1924, as amended by Government Notices Nos. 2015 of 1925, 112 of 1929 and 1456 of 1933.)

Paragraph 13 (i) reads:—" An incision shall be made into each shoulder behind the elbow, except in the case of a carcass intended for export overseas. In the case of a pig carcass intended for bacon an incision shall be made in the fillet (psoas muscle) in lieu of the aforesaid incision."

Paragraph 16 (i) reads:—"Every meat inspector finding evidence of bladderworm disease (measles) in a slaughtered animal during examination in accordance with Regulation 12 (General examination for all carcasses) and 13, shall further make the additional examination of:—

Head: Inspection incisions into inner and outer muscle of the jaw.

Tongue: Inspection of the surface and incisions into the muscles of attachment and the tongue proper.

Pluck: Examination of the heart and oesophagus.

Stomach and Intestines: Examination of the outer surface of the stomach and intestines.

Carcass: The following inspection incisions shall be made into *each* side of the carcass:—

Muscles of the shoulder behind the elbow ...	7 incisions.
Chuck (by which is understood the muscles on the dorsal aspect of the thoracic cavity)	1 incison.
Muscular diaphragm	2 incisions.
Fillet	3 incisions.

Apart from the foregoing, three incisions must be made into the pillars of the diaphragm."

It will be observed that according to the above regulations the meat inspector is virtually allowed to make only one incision into each shoulder behind the elbow, or in the case of bacon pigs, into the psoas muscles in lieu of those incisions, and three incisions into the pillars of the diaphragm. He is only entitled to make the subsequent incisions enumerated in paragraph 16 (i) after he has found evidence of bladderworms in the routine incisions.

Cysticerci cellulosae are occasionally encountered in most unusual locations, and may very easily be overlooked in routine inspection. In September, 1935, I reported an interesting case in the *Journal of the South African Veterinary Medical Association*, 6 (3), p. 191. The senior meat inspector at Bloemfontein abattoir, on examining the submaxillary lymphatic glands of a pig for tuberculosis, found a *cysticercus* in the gland of one side. I confirmed his diagnosis of *C. cellulosae*. In none of the legal routine incisions were *cysticerci* found, but on making the secondary incisions laid down by paragraph 16 (i), *Cysticerci cellulosae* were found in the following locations:—

In 7 incisions into the M. Triceps Brachii and M. Deltoideus on each side 2 *cysticerci*; Psoas muscles 1 *cysticercus*. No *cysticerci* were found in any other incisions. It is plain that but for the fortunate discovery of a *cysticercus* in the submaxillary lymphatic gland, a very unusual site, the carcass would have been passed as fit for human consumption.

In November, 1936, Dr. Bekker, Municipal Veterinary Officer, Pretoria, encountered several *cysticerci* in a pig's liver. No *cysticerci* were found in routine incisions . Being in doubt as to whether these bladderworms were *C. cellulosae* or *C. tenuicollis*, Bekker sent the liver to Dr. Mönnig, Onderstepoort. In answer to an enquiry from me as to the identity of the *cysticerci*, Dr. Mönnig replied:—" The bladderworms in the pig liver sent by Bekker are unfortunately not fully developed. The hooks are still young and imperfect, and so it is not possible to identify them, but the number of hooks coincides with that of *C. cellulosae*."

For economical reasons it has been customary in most South African abattoirs to condemn all pig carcasses found infected with measles, although instructions in Paragraph 16 (2) and (3) do not preclude lightly infected pig carcasses from receiving similar treatment to lightly infected bovine carcasses in cold storage at *minus* ten degrees Centigrade for 14 days.

In Bloemfontein, therefore, we were afforded the opportunity of studying predilection sites in thirty lightly infected carcasses, which were minutely dissected.

Measles were found:—

In muscles above the elbow only (Triceps, etc.) ...	10 cases.
In muscles above the elbow, plus psoas	4 cases.
In muscles above the elbow, plus psoas, plus thigh muscles	3 cases.
In muscles above the elbow, plus thigh muscles ...	5 cases.
In muscles above the elbow, plus cervicals	2 cases.
In muscles above the elbow, plus heart	1 case.
In muscles above the elbow, plus tongue	1 case.
In muscles above the elbow, plus tongue, plus cervicals	1 case.
In heart only	1 case.
In muscles above the elbow, plus psoas, plus submaxillary lymphatic gland	1 case.
In heart, plus tongue, plus oesophagus, plus cervicals	1 case.

In other words, in 30 specially observed lightly infested carcasses at Bloemfontein, *cysticerci* were found:—

In muscles above the elbow in	28 cases.
In muscles of the thighs in	10 cases.
In psoas muscles in	8 cases.
In muscles of the neck in	4 cases.
In the heart in	3 cases.
In muscles of the tongue and tongue itself in	3 cases.
In muscles of the oesophagus in	1 case.
In submaxillary lymphatic gland in	1 case.

According to these observations the muscles above the elbows must overwhelmingly be accepted as the commonest predilection site. In only two cases in the 30 pigs which had less than 10 measles in the ordinary routine incisions, did we not find measles in that location.

In grossly infested carcasses we observed bladderworms in the following organs:—

> Liver three occasions; kidneys twice; brain six times; eyeball twice; testicle once; vagina and uterus once; stomach and exterior of the intestines once. In all those cases infestation was very heavy, and the commoner sites were "swarming" with measles.

To summarize, judging from our observations at Bloemfontein, I regard the order of frequency of infestation in the various parts of the pig's carcass to be:—

1. The fore-quarters above the elbows (shoulder of pork).
2. The hind limbs above the hocks (leg of pork).
3. The psoas muscles and muscles on the ventral surface of the vertebrae.
4. The cervical muscles and the intercostals.
5. The tongue and its muscles.
6. The heart and the perineal region.
7. The oesophagus and the diaphragm.
8. The muscles of the face and the abdominal muscles.
9. The brain.
10. The liver, fat and superficial fascia.
11. The eyeball, conjunctiva, etc.
12. Sexual organs, and internal organs not mentioned above, also lymphatic glands.

Von Ostertag (1913) gives the following predilection sites:—Abdominal muscles; muscular portion of the diaphragm; lumbar muscles; tongue; heart; muscles of mastication; intercostal muscles; cervical muscles; the gracilis and the sternal musculature. He mentions that the heart and the brain should be named as frequent locations for the hog bladderworm.

Kukuljevic (1906) mentions that on four occasions he found *Cysticerci cellulosae* in the eyeball of pigs, and more frequently in the brain.

Hutyra and Marek (1916) mention that *Cysticercus cellulosae* is sometimes found in the spinal cord. They mention the following order of frequency:—Deep muscles of the shoulder and chest; abdominal muscles; nape and neck muscles; diaphragm; intercostals; adductors of the thighs; less frequently in the muscles of the tongue and the heart, "and in very severe cases in the brain, eyes, liver spleen, lungs, lymphatic glands and fat."

Vosgien (1911) found the following order in the pig:—"The muscles of the chest, diaphragm, tongue, heart, muscles above the elbows, and less frequently the intercostals, psoas, masticatory muscles and muscles in the vertebral region."

At Boeleleng (Dutch East Indies), in 1927 Le Coultre found the order of frequency of infestation in 29 pigs to be:—Muscles of mastication; shoulder and the muscles of the upper arm; psoas muscles; neck muscles; vertebral muscles; tongue muscles; abdominal muscles; muscles of hind legs below the patella; muscles of fore legs below the elbow; intercostals; and lastly came the diaphragm, heart and brain.

The Japanese workers Eguchi and Nishiyama (1930) found the predilection sites in infested pigs in Prefecture Okinawa to be:—Skeletal muscles and heart, and then in the brain, orbit and cheeks. Out of 42 infested pigs they found measles in one case in each of the following locations:—Spleen, kidneys, stomach, intestinal wall.

Hertwig (1885) mentioned that *Cysticerci cellulosae* were rarely found in the liver, lungs, spleen and kidneys.

Irvine-Smith (1910-11) advised the Johannesburg Municipal Council at that time, in his annual report that "the following parts of pig carcasses are closely examined:—Tongue and heart; muscles of the neck; breast; intercostals; midriff and psoas."

Degeneration of Cysticercus cellulosae.

Quite frequently degenerated *cysticerci* may be found in pigs, although more rarely than is the case with *Cysticercus bovis* in beef.

Von Ostertag states that degeneration usually occurs at an early developmental stage. At Bloemfontein we frequently observed degenerated measles in old pigs, although on one occasion I noticed both caseous and apparently viable *cysticerci* in a pig of about 12 months old. This observation differs from that of von Ostertag, who states (1934) that all parasites in the case of *C. cellulosae* are affected (simultaneously?) by the process of decay, contrary to the rule in *C. inermis* (*bovis*).

Degeneration of the *cysticercus* takes the form of caseation and calcification—progressive stages of the same degeneration. The fluid contents of the cyst progressively dry to caseation and ultimately to calcification. Progressive atrophy of the cyst itself occurs, and the shape of dead *cysticerci* is affected. Dead *cysticerci* appear as elongated, sometimes slitlike structures which, occasionally may be barely visible to the naked eye, appearing as white specks, and sometimes they may be the size of hemp seeds. During the caseous stage of degeneration the pig measle may appear gray in colour, but when calcified it is usually pure white.

Neumann states that it is usually age that brings about degeneration of the *cysticerci*, and their transformation into small, round, hard and compact grains, impregnated with calcareous matter and destitute of fluid. The pork butchers (in France) name the disease *dry measles*. Neumann adds that the degeneration of the *cysticerci* is centripetal, that is, it begins with the external membrane and finishes with the scolex; and this is most evident in caseous or pseudo-purulent degeneration. When numerous *cysticerci* have degenerated, the heart and skeletal musculature are found to be sprinkled with white granules. (Neumann.)

Under the microscope, a tough connective tissue membrane and a more or less strongly calcified centre may be demonstrated in the calcified structure. (von Ostertag.) Sometimes demonstration of the classical calcareous corpuscles and hooks in a degenerated cyst may serve as a diagnostic feature of the former bladderworm.

SYMPTOMS IN THE PIG.

Clinical symptoms of *Cysticercus cellulosae* infection are extremely rare in the pig. The severe constitutional disturbances sometimes met with in the human infection seldom reveal themselves in pigs.

MacArthur (1934), in discussing the incidence of human cysticercosis in the British Army, mentions the prolonged period after initial infection, before constitutional disturbances occurred in patients he studied. He mentions the fact that the parasites may be present in the human body for several years (e.g. six to eleven years), before cerebral symptoms become apparent. In the case of the pig, in applying MacArthur's remarks on the human disease, it is less likely that cerebral symptoms will develop, since it is seldom that a pig will be allowed to live to that age before slaughter. MacArthur also states that it is his belief that *cysticerci* while alive, usually enjoy a relative tolerance on the part of the host, but that after their death they act as foreign irritants and bring about the degenerative changes in the tissues of the human host. He makes it clear that severe pathological changes of the infected tissues only appear a number of years after initial infestation.

Some of the older writers, however, have recorded severe constitutional disorders in pigs. Gréve, according to Neumann, reported that he noticed in many measly pigs an increased sensitiveness in the snout, which prevented their burrowing. In eating grain off a hard floor they avoided contact with that part as much as possible, by raising the nose and upper lip and prehending the food with their tongues. Tapped slightly on the end of the nose with a stick, the measly pig squealed with pain, while a healthy pig would remain indifferent. Very measly pigs had the snout more or less soft and flaccid.

In a pig suffering from gross infestation with measles, Sabotta (1880) observed complete paralysis of the tongue, which was invaded by *cysticerci*. The prehension of food was, therefore, impossible, " and the animal perished from inanition ". Florman (1819) saw a very manifest turning round in circles in a case where *cysticerci* were located in nerve centres. Rehrs (1842) witnessed epileptiform convulsions, grinding of the teeth, ptyalism and rabid-like vertigo. At the autopsy Rehrs found in the cerebrum and cerebellum an enormous number of *Cysticerci*, "several of which were of exceptional size". Rabid-like symptoms were also noted by Foucher (1874). Vertigo and a form of blindness in which case the brain was softened and contained more than a hundred *cysticerci*, were observations made by Neubert (1861). Lippold (1875) had a case in which the pig died after presenting all the symptoms of encephalitis. On post-mortem twelve *Cysticerci cellulosae* were found in the pia mater.

Neumann mentions that in chronic and generalized infections pigs may be feeble, easily put out of breath, have difficulty in following the herd, may later develop diarrhoea, foetid breath, prostration, then death.

Hutyra and Marek mention that in very severe infestations similar symptoms to those described above, may appear, and in addition hoarseness may result owing to involvement of the laryngeal muscles. (It is perhaps possible that in some of these cases, the pigs were also infested with *Trichinella spiralis*, which is known to cause muscular weakness and particularly hoarseness.)

Daubney (1936) mentions a case which, quite recently, was brought to the Veterinary Research Laboratory, Kabete, Kenya Colony. The affected animal was fevered and showed all the clinical symptoms of acute muscular rheumatism. It experienced considerable difficulty in rising, and any movement or manipulation occasioned pain. Post-mortem, the carcass was found to be grossly infested with viable *Cysticerci cellulosae*, and the infestation, according to Daubney, "had undoubtedly been responsible for the clinical manifestations".

Clinical symptoms are not likely to be noticed at abattoirs, since pigs are generally slaughtered within twenty-four hours of their arrival. In practice no cases showing clinical symptoms of constitutional derangement were observed at Bloemfontein, although in some dressed carcasses infestation was so heavy that barely spaces of 5 mm. could be found between the measles. It was, however, noticed that on rare occasions the flesh of heavily infested carcasses had a pale colour and was slightly dropsical.

In living cases the disease is generally recognised only in cases where the parasites are situated in a visible mucous membrane, e.g. in the conjunctiva of the eye, or in the lens. Externally *cysticerci* may sometimes be seen or felt with the hand in the tongue, mostly at the edges on the under surface, or in the fraenum linguae. Sometimes also *cysticerci* may be felt in the folds of the rectum, or in the anal ring. In heavily infested pigs *cysticerci* may be seen "bubbling" out of the perineal region, with the first incision of initial dressing of the carcass, but ante-mortem they are difficult to palpate in that region. Inspection of the tongues of pigs for measles has been practised since the days of Aristophanes, was commonly practised in Germany and in France in the Middle Ages, and is practised in most countries, where porcine cysticercosis is a common disease, at the present time. Tongue inspection is a common practice of South African farmers, some of whom consider themselves experts and have actually told me that only a certain percentage of their pigs reach the Bloemfontein Abattoir, since they withold all pigs found to be measly by means of the tongue inspection, and they sell such pigs to their natives, instead of providing us with so much material for our by-products department.

The South African method of inspection consists of the following: The pig is thrown onto its side. Taking advantage of its squeals, a stout stick or plank is forced between the jaws, and with his hand wrapped in a towel or cloth, the farmer grabs the tongue pulls it out of the mouth and examines it. His native attendants, meanwhile, use the stick as a lever to hold the mouth open. The Serbian method of inspection, according to Kukuljevic (1906). was almost identical to the South African method.

That measles can only, with certainty be diagnosed in very grossly infested cases, is shown by the following observations conducted at Bloemfontein Abattoir, where we instituted a three months' inspection of living pigs' tongues. During that period exactly 25 per cent. of the total measly pigs slaughtered showed *cysticerci* in their tongues.

NATURAL INFECTION OF THE PIG.

The pig is naturally a scavenger and burrower, and may almost be termed omnivorous. When not confined to a sty, its natural instincts are to haunt the precincts of rubbish heaps, manure and excretum dumps, latrines and the dirtier parts of the farm-yard. In the more primitive parts of South Africa, among European as well as native habitations, pigs frequently have the run of the farm-yard, and may even enter the kitchen or native huts. On many farms latrines or privies are neither provided for Europeans nor natives, and in the vicinity of native huts, or in the rural locations and reserves such commodes are quite unknown. The primitive and unhygienic farmer, and nearly all natives will walk barely a hundred yards from the homestead in order to perform their natural functions. That type of farmer (fortunately becoming more scarce) or native very rarely takes the trouble to sty his pigs. The only feed which is provided for the unfortunate pigs consists of an occasional ration of pumpkin, a few mealies, potato peels and other rubbish from the kitchen, and further the pigs must forage for themselves. The most natural result is that the pig will follow its owner and act as an " efficient scavenger ". It is well known that among many of our so-called " Poor Whites " this mode of scavenging is encouraged. I was once told that privies on farms were unnecessary evils, since they stank and encouraged flies. That particular " farmer " and his entire household used the rear of a quince hedge close to the homestead, since it was far " cleaner and the pigs cleaned up everything ".

In the western Cape pigs are frequently driven to pick up acorns under the beautiful old oaks on the farms. Unfortunately, at the same time the farmers' Cape Coloured servants select the shade of those oaks as lavatories. Heavy infestation among Cape pigs frequently follows.

Le Coultre (1928) relates a similar state of affairs among the native Balinese. He picturesquely describes the remarks of an old Headman who stated that he merely whistled for his pig to come and clean up his excretum. Except for the fact that the primitive ones among our South African farmers and natives have perhaps not trained pigs to follow their whistles, there is very little difference between our most primitive farm hygiene and that of Bali.

Undoubtedly a similar state of affairs must exist in the more primitive parts of Russia, Serbia, Lithuania and other parts of Europe, where the incidence of *C. cellulosae* is still high.

Commenting on the usual heavy nature of infestation in pigs, Veenstra (1921) asks the following questions: "Does a pig generally become more heavily infested than a bovine? Or do the eggs develop more readily in the pigs' bodies than in those of bovines? Or do more *T. saginata* eggs get lost into the soil?" He then adds that pigs are less particular than bovines in picking up their food. Veenstra's queries can be elucidated by the fact that proglottides of *T. solium* are voided in chains, seldom singly, and although the majority of the eggs may escape from the proglottis, the contact intestinal faeces of the human host must, therefore, probably carry more *T. solium* eggs than would be the case with *T. saginata*, where segments are voided singly and sometimes spontaneously. In other words, more *Taenia solium* proglottides become gravid and "ripen" at a time than those of *Taenia saginata*. It stands to reason, therefore, that the voracious pig, in ingesting, as it commonly does, an entire human stool, will take in many thousands of ova.

Infection of the pig with *C. cellulosae* can only result from the ingestion of ova of *T. solium*, most frequently obtained by the ingestion of entire stools, or from the ingestion of excreted gravid proglottides in which ova may be present.

Unfortunately, infection of pigs belonging to scrupulously particular farmers may occasionally result, owing to the wantonness of their native swine-attendants. Three such instances occurred in the Bloemfontein District within a few weeks of each other, during the past year. Each of the three farmers could be classed among the foremost pig breeders in the Province, and when measles were found among a number of their respective pigs, their faith in the theory of the mode of infection was rudely shaken. One of them told me he could not believe it, since his pigs were scrupulously stied and fed, a fact of which I had already full knowledge.

I told each farmer that I was sure one of his native staff had been relieving himself in his sty, and advised them to keep a careful watch. It was not long after, when two of the three farmers enthusiastically told me that each had caught a native, one of whom actually carried tapeworm, in the act. What those two farmers did to their respective native culprits was not mentioned, but can well be imagined!

According to Gerlach infection of the pig occurs more readily in the young subjects, e.g. up to half a year old. On the other hand there are not many records of infection in sucking pigs. The reason for this may be that sucking pigs remain with the dam, and their diet consists mainly of milk. They are, however, not scavengers. Unlike infection of the bovine with *C. bovis*, intra-uterine infection of foetuses with *C. cellulosae* has seldom or very rarely been recorded. Hervieux (1838) recorded a case in sucking pigs. " The sucking pigs were found affected with measles—two in a litter of twelve." He further hinted at prenatal infection: " A sow that was reared by the writer, was mated to a very healthy boar, and the former bore six measly sucklings ".

At Bloemfontein our record of the youngest measly pigs was 10 weeks. In December 1934 we found four out of a consignment of six small sucking pigs, 10 weeks old, to be heavily infested with mature viable measles.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF C. CELLULOSAE IN PORK.

In ante-mortem cases, except for the examination of the tongue already described, there is no practical method of diagnosis. Serological tests have been tried, but are not specific and are not practical under ordinary conditions. Sparapani (1915) tried precipitin tests.

On post-mortem examination, or in practical meat inspection, there is really only one parasite which may be confused with *C. cellulosae*, viz. *C. tenuicollis*, the immature stage of *T. hydatigena* (*marginata*) of the dog. As a general rule the latter *cysticercus* does not develop in muscles, and is most frequently found under the large serous membranes, the peritoneum and pleura, and chiefly in the omentum, mesentery and liver. Its size may vary from that of a pea to that of a tennis ball. When situated in the parenchyma of the liver, it may be confused with *C. cellulosae*, since its size is restricted, seldom exceeding that of a pea Older text-books and writers maintained that *C. tenuicollis* was never found in muscle fibres. Recently Mönnig (1934), referring to some American literature, stated that " *Cysticerci* (*tenuicollis*) developing in muscles may not be so large and have possibly sometimes been mistaken for *C. cellulosae* ". Larger vesicles, e.g. those found under serous membranes, on the omentum, etc., are filled with fluid, may be quite flaccid, and are hardly likely to be mistaken for *C. cellulosae*. These larger *cysticerci* evaginate the scolex very easily, and reveal a scolex with an unusually long neck, from the end of which hangs the flabby bladder. The evaginated scolex may be studied and will reveal a rostellum bearing two rows of hooks numbering from 26-44 (cf. *C. cellulosae* 22-32). The larger hooks are 0·17 to 0·22 mm. long (cf. *C. cellulosae* 0·16 to 0·18 mm.) and the smaller hooks 0·11 to 0·16 mm. (cf. *C. cellulosae* 0·11 to 0·14 mm).

The hooks of *C. cellulosae* are more curved (sickle-shaped) than those of *C. tenuicollis* (scythe-shaped). According to Von Ostertag, the root process of the smaller hooklets of *C. cellulosae* has no cleavage as is found in that of *C. tenuicollis*. In the 1913 edition of von Ostertag's " Handbook of Meat Inspection " mention is made that " Schwarz examined 1,000 specimens each of *C. cellulosae* and *C. tenuicollis*. He noticed that in *C. tenuicollis* as a rule one or more small hooklets were demonstrable, the basal process of which was bifurcated. In the thousand specimens of *C. cellulosae* examined by Schwarz this was not the case in a single instance. Reissmann has confirmed these observations ". In his 1934 " Text-book of Meat Inspection ", however, von Ostertag mentions that the small hooklets of *C. tenuicollis* are characterized by their bifid form, " but it must be noted that in the small hooklets of *C. cellulosae* there is also a division through a median furrow ".

Manegold (1931) showed the tremendous variations between the number of hooks, predilection sites and size of *C. cellulosae, C. tenuicollis* and *C. ovis* quoted by different text-books and authorities. In order to establish a differential diagnosis he found that 32 hooks were the commonest in 500 *C. tenuicollis* scolices, not 36-38 as was frequently quoted in many text-books. In the majority of cases (95·6 per cent.) 28-36 hooks were found.

It is very improbable that *C. cellulosae* will be confused with any other pathological conditions, but the following have been mentioned in the discussions on differential diagnosis:—

(*a*) *Echinococcus cysts.* These either have no scolex (sterile form), or numerous brood capsules, each with many scolices. The cuticle of the *Echinococcus* is thick and concentrically laminated.

(*b*) Calcified *C. cellulosae* may (very improbably) be confused with calcified *Sarcospordia-Sarcocystis miescheriana.* These parasites may be about 4 mm., long by 3 mm. wide, readily undergo calcification, and are the commonest form of calcareous concretion in the muscle fibres of swine. They are especially found in the abdominal muscles and the diaphragm. (They are more likely to be confused with *Tirchinella spiralis.*) In uncalcified specimens the sickle-shaped sporozoites may be demonstrated.

(*c*) Small actinomycotic nodules may possibly be taken for *Cysticerci cellulosae.* In actinomycosis radiation of the mycelium may be demonstrated microscopically.

(*d*) Tuberculosis of lymphatic glands may be confused with caseous *cysticerci* in those locations. Microscopical examination will settle the diagnosis.

Dead (caseous) *C. cellulosae* may be identified *microscopically* by demonstration of the characteristic calcareous corpuscles and the nature of the hooklets.

Vosgien (1911) mentions two methods of identifying *C. cellulosae* in sausages and other minced meats. The first is that of Schmidt-Mulheim, in which the product is warmed to 40° C. in 6 to 8 times its volume of a 1 in 122 hydrochloric acid solution. *Cysticerci* with hooks resist this treatment and become visible. The second is that of Rissling, who employed a soda solution.

INFECTION WITH CYSTICERCUS CELLULOSAE OF OTHER ANIMALS.

The only other animals in which infection with *Cysticercus cellulosae* need be discussed are man, the dog and the monkey. Human cysticercosis will receive consideration later in this work.

Infection in the dog has generally been accepted as a scientific fact, whereas until 1936 few authentic cases in the monkey were recorded. In 1936 Walker recorded what he believed to be a definite case of *C. cellulosae* in a monkey. He mentions that the monkey is cited in many scientific books as an intermediate host of *Taenia solium* but in actual fact the condition in that animal has extremely rarely, authentically, been found. Medical literature during the past 50 years has been silent on that subject, and the only other records which Walker could find were four cases mentioned by Vosgien, the one found in the eighteenth century, and the other three in the nineteenth century.

Relating the case history of his subject, Walker reminds us that the exact incidence of infection in monkeys will not be known until post-mortem examinations are systematically performed on large colonies of monkeys, over a prolonged period of time. His subject was an immature *Macaca mulatta*, bought from an eastern animal dealer. Enquiry from the dealer led to the information " that the animals in that lot were from Lucknow, that several had worms, got very skinny, and eventually died ". Apparently the type of worm was not investigated. A large flap of bone was turned down on the left side of the skull, with the intention of stimulating the cerebral cortex, but upon opening the dura mater " quite unexpected pathology was found ". " Both in the subdural and subarachnoid spaces were numerous cysts ranging in size from 3 mm. to 15 mm. Those in the subdural space were so loosely attached that when the dura was opened they fell out. Even those in the subarachnoid space enucleated readily when the arachnoid was nicked." On close examination Walker saw several cysts within the brain substance, partially covered by the cortex. After this he killed the monkey and did a complete post-mortem examination. He found that practically every muscle in the body contained one or more cysts. He gives the following description of his obervations: " The muscles of the back had many; those of the extremities likewise were studded with cysts. Even the intercostal muscles and the diaphragm had cysts. Two cysts were found within the heart muscle. The liver, spleen and both kidneys contained typical cysts. There was no evidence of any primary worm from which the infection may have arisen."

Walker then proceeds to describe the microscopical features of the scolices of the bladderworms. He points out that the severe pathology of human cysticercosis was not observed in the monkey. When the scolices were examined under the microscope " four suckers, surrounding the rostellum with a number of hooklets " were seen. The actual number and the characteristic features of the hooklets were not mentioned, and this fact may be cited as the main argument against the diagnosis of Walker, who maintains that he had dealt with a typical case of *C. cellulosae*. He, somewhat inconclusively, unfortunately, claims that " the presence of both suckers and hooklets on the scolex serves to define the larva as that of the *Taenia solium*."

Nevertheless, Walker has apparently been the first author in the last 50 years to have described a case, which to all appearances may be accepted as a case of generalized *Cysticercus cellulosae* in a monkey.

If Walker's diagnosis is scientifically correct, it is quite possible that, as he has pointed out, the incidence may be higher than has been anticipated among the various species of monkeys. In South

Africa it has been noticed that most tamed monkeys are scrupulously particular of their food. In the wild state it may be possible that monkeys will eat human excretum, especially if other normal foods are scarce, and ova of *Taenia solium* may be consumed by the ingestion of contaminated roots, herbs, etc. It is also not at all unlikely that human beings may contract *Taenia solium* through eating measly monkey flesh, since in South Africa several native tribes, e.g. the Amaxosa and some Bechuanas are very fond of monkey flesh.

It is an old accepted fact that *Cysticercus cellulosae* does, with varying incidence, occur in the dog. No doubt the incidence of infection with *Taenia solium* among humans, as the result of eating dogs' flesh only occurs in eastern countries, but universally dogs are susceptible to infection with *Cysticercus cellulosae* as the result of ingestion of human excretum containing ova of *T. solium*. As far as is known only two cases of canine cysticercosis have been observed in South Africa. Two brains of dogs suspected of rabies were found at the Onderstepoort Laboratory to contain many *C. cellulosae*. These brains were forwarded by Field Veterinary Officers to be examined for rabies, but both were negative for that disease.

Interesting statistics may have been brought to light if it had been possible to hold autopsies on all dead roaming dogs, especially the so-called "Kaffir dog" variety and scavenging and marauding farm dogs. It obviously does not follow that because in countries where dogs' flesh is not eaten, and man will not contract *Taenia solium* from that source, the converse infection of the dog with *C. cellulosae*, through eating infected human excretum may not result. Walker's remarks, therefore, regarding the possible fairly high incidence of cysticercosis among monkeys are equally applicable to dogs, if a thorough survey could be made in parts of Europe and America, Asia and Africa, Continents in which *T. solium* is still fairly common.

It is quite possible that the early Phoenicians, who ate no pork, but were very fond of dogs' flesh, may have contracted *Taenia solium* from measly dogs' meat.

Poisson (1930) recorded a case of *C. cellulosae* in a dog in Madagascar.

Undoubtedly, clinical symptoms are more apparent in dogs than in pigs, and cases resembling human cysticercosis, with its accompanying cerebral, nervous and ocular symptoms have been recorded by a number of writers. The main reason for this may be attributed to the fact that in general only such dogs which have shown rabid-like signs, epileptiform symptoms, blindness, etc., have been autopsied, and for every one of those cases, many hundreds of cases of ordinary intra-muscular cysticercosis may have passed unobserved, that is, the dogs have eventually died natural deaths and been buried or discarded into the rubbish bin, without further examination. On the other hand, in cases which have definitely been autopsied, cysts have most commonly been found in the brain in dogs, and less frequently in the conjunctiva, in the eyeball, sub-retinal, and in the general intramuscular tissue. From cases actually observed, the brain may, therefore, be cited as a "predilection site" in the dog.

Among many others, Ball and Marotel (1903), Lesbre (1882), Repiquet and Salvatori (1906) and Van der Slooten (1892) recorded cases of canine cerebral cysticercosis. Rivolta (1865) found cerebral cysticercosis in a dog which died suddenly from epilepsy, without having shown any evidence of previous illness.

Vogel (1870) autopsied and found *Cysticerci cellulosae* in the eyes of a dog which had gone blind.

Siedamgrotsky (1871) recorded a case which was suddenly seized with cramps and convulsions, especially of the jaws; " then it had fever, prostration, accompanied by vertigo and delirium, and death occurred during the day; 23 *cysticerci* were found lodged in the superficial part of the two cerebral hemispheres; nothing abnormal was observed elsewhere." Lesbre (1882) described a case in which the dog had been paralysed for two days, but for a long time previously it had " grinding of the teeth, was excited, and had attacks of vertigo ". On post-mortem 30 to 40 *Cysticerci cellulosae* were found in different parts of the brain.

Generalized intra-muscular cysticercosis was described by Dufour and Gacon (1889), who found *cysticerci* in the neck muscles, the tongue, the general musculature, the heart and the lungs. Leblanc and Megnin (1873) found *cysticerci* in the neck, the liver and the pancreas. Suffran (1909) found a number of small swellings in the skin of a four years old fox-terrier dog. On microscopic examination the swellings proved to be *C. cellulosae*. Trasbot and Railliet (1887) examined numerous canine *cysticerci* and found that they were identical with those of pigs. They confirmed the fact that dogs were hosts of *Cysticercus cellulosae*.

Most recent reports of cases of canine cysticercosis cellulosae originate from Asia.

Rao (1933) writes that *C. cellulosae* occurs in the pig and in the dog in the Madras Presidency of India.

Meyer (1933) records that dogs are frequently used as food animals in the Bataklands, Residency Tapanoeli, Dutch East Indies. In the Sub-division of Toba dogs are slaughtered in the abattoir and the flesh is sold in bazaars. Whilst inspecting dog carcasses for *Trichinella*, Meyer found *Cysticerci cellulosae* in a dog. The cysts were mainly in the heart muscles and had the same appearance as those in pigs. Later he found four more cases. He points out that in that part of the Dutch East Indies dogs eat human excretum as readily as pigs do.

Bergeon (1928) reports cases of cysticercosis in dogs in Hanoi (Tonkin). In 1919 he first discovered *cysticerci* to be the cause of rabid-like symptoms in a ten-years-old dog. After that he caused all dog carcasses to be examined in their " Section ", and found 138 cases of *Cysticercus cellulosae* between 1919 and 1924. Bergeon accounts for the high incidence of canine cysticercosis by the frequency of taeniasis among the Tonkinese. The natives are readily infected with *Taenia solium* owing to their ~~habit~~ of eating almost raw dogs' flesh, which has been lightly smoked over a straw fire. All Bergeon's cases were *C. cellulosae* and no case of *C. bovis* was found in dogs, although *T. saginata* is relatively common among the natives. Bergeon recommends that meat inspectors in the Far East should carry out systematic inspections of all dog carcasses slaughtered.

C. Infestation of the Bovine with Cysticercus bovis.

As far as scientific investigations have gone, the *Cysticercus bovis* has only been found in the bovine and, very rarely, in man. Older writers have recorded measles resembling as they thought, *C. bovis* in antelopes, deer, etc., but they were probably mistaken, and very likely the *cysticerci* they encountered were armed forms, e.g. the *Cysticercus cervi* in deer, or kindred forms.

Practically and scientifically we may, therefore, regard the bovine as the only domesticated animal which harbours the intermediate stage of the *Taenia saginata*.

Infestation in the adult bovine is usually of a very light nature. Most observers, throughout the world, have in routine inspections encountered but a few cysts in infected carcasses, or in the usual inspection incisions, and, exceptionally cases of gross or light generalized infestation have been met with. The present author has never yet come across a case in which infestation in any way resembled, in severity, that of a grossly infested pig carcass with *C. cellulosae*. Neumann, however, quotes J. Fleming, who counted 300 living *cysticerci* in a pound of psoas muscle.

PREDILECTION SITES.

Owing to the nature of bovine infestation, it is justifiable to regard certain locations as "predilection sites". It is quite impossible to incise a bovine carcass at random, and, for that reason, Regulations lay down certain incisions in which *cysticerci* are frequently found. The incisions are to be made into muscle groups where as little mutilation of the carcass, as possible, will result.

Between 1st May, 1936, and 31st January, 1937, twenty-five bovine carcasses were totally condemned at the Bloemfontein abattoir. By minutely dissecting these carcasses, an opportunity was afforded to study the predilection sites, especially in those portions of the carcass which usually escape incision. These operations entailed a good deal of time and work, but it was felt that a true and representative survey of the most common sites of infestation could only be made by carving a series of condemned carcasses into as thin slices as possible.

It is admitted that a more comprehensive idea and summary would be formed only after about a hundred or more carcasses are so treated, but, on the other hand the ratio of light infestation to heavy infestation in South Africa is about 10:1, and grossly measled material in an abattoir of the size of this, is relatively scarce. In addition to the observations in the 25 heavily infested carcasses, records were made in 113 consecutive lightly infested carcasses.

Le Coultre, during his observations in Bali in 1927, was afforded the unique opportunity of boning every infected carcass, and thus studying predilection sites. Under European conditions we are precluded from emulating Le Coultre's investigations.

A fairly comprehensive idea of the most commonly infested muscle groups may be obtained by reference to the subjoined table, which shows the number of measles which we found in each group, in 25 carcasses at Bloemfontein.

Carcass Number.	Total Measles.	(1) External Masticatory	(2) Internal Masticatory.	(3) Tongue.	(4) Oesophagus.	(5) Heart.	(6) Diaphragm.	(7) Sternal Muscles.	(8) Ext. Thorax and Intercost.	(9) Cervicals and Hump.	(10) Ext. Muscles of Vertebrae.	(11) Shoulders and Elbow.	(12) Extensors and Flexors of Carpus	(13) Psoas.	(14) Hind Limbs.
1	30	3	2	—	—	—	1	—	2	5	3	6	—	—	8
2	31	4	1	—	—	1	—	—	—	3	4	6	1	2	9
3	33	—	1	2	—	1	—	—	—	—	5	8	2	—	14
4	91	12	3	—	2	—	2	—	6	5	8	22	4	1	26
5	47	7	4	1	—	—	—	—	3	2	4	16	—	—	10
6	38	11	3	—	—	—	—	—	5	—	—	8	3	—	8
7	73	6	—	9	—	2	—	—	—	16	—	22	4	—	14
8	19	3	1	—	—	1	—	3	—	—	2	4	—	—	5
9	24	7	—	—	—	—	—	—	—	3	4	3	—	2	5
10	42	6	2	—	1	—	—	1	2	—	2	9	3	—	16
11	328	29	3	12	22	7	12	—	—	15	7	101	16	12	92
12	64	16	3	—	—	—	—	7	2	5	—	17	—	-	14
13	21	3	2	—	1	1	—	—	1	2	—	7	—	—	4
14	29	9	1	4	—	—	—	1	—	—	—	12	—	—	2
15	44	7	—	2	—	2	—	—	1	5	3	16	—	—	8
16	40	5	2	1	—	—	1	2	—	3	2	8	—	2	14
17	49	3	—	—	—	—	—	—	2	4	2	23	2	—	13
18	105	17	3	—	2	3	2	—	6	9	5	27	—	—	31
19	37	6	2	1	—	1	—	2	3	5	—	8	6	—	3
20	73	13	—	—	—	—	—	4	—	7	—	31	2	—	16
21	70	6	3	—	—	—	—	—	—	3	7	19	—	4	28
22	44	7	2	—	—	—	—	1	—	1	2	14	1	—	16
23	85	3	2	—	—	2	—	—	—	3	8	26	—	2	39
24	53	13	1	—	—	—	—	—	—	1	1	7	11	—	19
25	28	3	3	—	—	—	—	—	—	1	3	2	3	1	12
TOTAL	1,498	199	44	32	28	21	18	21	33	98	72	422	58	26	426

Judging from the foregoing table, in 25 heavily infested carcasses, it would appear that the hind limbs harbour the most parasites.

Out of a total of 1,498 measles found in the 25 animals—

(14)426 measles were found in the hind limbs in 25 animals;

(11)422 measles were found in the shoulder and elbow in 25 animals;

199 measles were found in the external masticatory muscles in 24 animals;

(9) 98 measles were found in the cervicals and hump in 20 animals;

(10) 72 measles were found in the external vertebrals, etc., in 18 animals.

(12) 58 measles were found in the extensors and flexors of carpus in 13 animals; 44 measles were found in the internal masticatory muscles in 20 animals;

(8) 33 measles were found in the external thoracic and intercostals in 11 animals;

32 measles were found in the tongue and its muscles in 8 animals;

28 measles were found in the oesophagus and its muscles in 5 animals;

26 measles were found in the psoas muscles in 8 animals;

21 measles were found in the heart in 10 animals;

(7) 21 measles were found in the sternal muscles in 8 animals;

18 measles were found in the diaphragm in 5 animals.

In two cases the total number of 433 measles was found, that is, 328 in one carcass and 105 in the other. A more representative average would be arrived at by subtracting the 433 measles from the total of 1,498, thus giving 1,065 measles in 23 carcasses, an average of 46·3 per carcass.

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(7) *Sternal muscles group* included superficial and deep pectorals.

(8) *External Thoracic and Intercostal group* included Posterior deep pectoral, Latissimus dorsi, Serratus ventralis, External abdominal oblique, and Intercostals proper.

(9) *Cervicals and Hump group* included Trapezius, Omo-transversarius, Upper Brachio-cephalic, Rhomboideus.

(10) *External vertebral muscles group* included Serratus dorsalis, Longissimus costarum, Longissimus dorsi.

(11) *Shoulder and Elbow group* included all muscles on the lateral and medial aspect of the shoulder and humerus up to elbow.

(12) *Extensors and Flexors of Carpus group* included Extensor carpi radialis, Extensor digitalis communis, and lateral and medial Digital extensor, and Superficial and deep digital flexors—all from elbow to carpus.

(14) In this group were included all muscles of the hind limbs.

It will be noticed that *cysticerci* were found in the shoulder and elbow and in the thigh muscles of all 25 cases. In 24 out of the 25 cases measles were found in the external masticatory muscles. The fact that less than half the number of measles was found in this group compared with the shoulder and the thigh, may be ascribed to the fact that in the latter pairs very large groups of muscles were dissected, whereas the sections of the masseters were considerably smaller. Statistics such as those shown in the table may, therefore, be misleading. I quite agree that the group of muscles known as the masticatory (internal and external) must certainly be considered as a most important predilection site, and in Bloemfontein it was extremely rare that no measles were found in this group in an otherwise measly carcass.

The analysis formed by our careful dissection has brought to light the fact that the hind quarters of beef are as important from the point of view of measles location, as the fore quarters. It is quite possible that numerous measly carcasses may escape detection because no provision is made in Regulations for the incising of any part of the hind quarter, except the psoas muscles, which, as our table shows, cannot be considered a very important predilection site.

In view of the comparatively frequent and heavy nature of infestation of the hind limbs, it was further decided to record the actual groups of muscles of the hind limb, in which measles were found, in the last six carcasses we dissected. The following was our recording:—

Carcass No. 20.—Sixteen measles in both hind limbs. Muscles Semimembranosus 4 measles.

Biceps femoris, lateral vastus, semitendinosus 8 measles.

Adductors 4 measles.

Carcass No. 21.—Twenty-eight measles in both hind limbs.

Gracilis, Adductors and vastus medialis group 12 measles.

Semimembranosus 1 measle.

Semitendinosus 5 measles.

Biceps femoris and lateral vastus 10 measles.

Carcass No. 22.—Sixteen measles in both hind limbs.

Gracilis and medial vastus 5 measles.

Vastus lateralis 2 measles.

Rectus femoris 1 measle.

Semitendinosus 4 measles.

Adductors 4 measles.

Carcass No. 23.—Thirty-nine measles in both hind limbs.

Vastus medialis, Pectineus and Quadratus femoris 21 measles.

Biceps femoris 1, lateral vastus and Semitendinosus 9 measles.

Adductors 9 measles.

Carcass No. 24.—Nineteen measles in both hind limbs.
Gluteus medialis 3 measles.
Gracilis and adductors 5 measles.
Semitendinosus 5 measles.
Gastrocnemius (fleshy belly of lateral head) 4 measles.

Carcass No. 25.—Twelve measles in both hind limbs.
Medial vastus 3 measles.
Biceps femoris, rectus femoris, vastus lateralis 7 measles.
Adductors 2 measles.

It will be noticed that measles were found in the adductors of all six carcasses (twelve hind quarters) observed. It must be emphasised that the adductors and most of the other large muscles on the medial aspect of the thigh should be considered very important locations for *Cysticerci bovis.* The large muscle groups on the lateral and posterior aspects of the pelvic limb are also important sites, but it would be impracticable to incise into them in ordinary meat inspection, without grossly mutilating the carcass. On the other hand the writer found that an incision parallel to and about an inch below the pelvic symphysis, deeply into the adductor muscle, did no material damage to the appearance of the quarter. The incision coincides, more or less, with those which butchers make in their shops in cutting up the steaks. The opinion of one of the local butchers was asked as to objections which might be raised against possible mutilation of the quarter by the making of such an incision, and he replied naively: "We will not object to the cut being made, but will be very annoyed if that will lead to your finding more measles". Practically, the incision is best made while the carcass is still on the floor, just after the abdomen has been opened and the pelvis cleft. If the incision is to be made after the carcass has been hoisted, meat inspectors will have to stand on ladders. When made on the ground, the cut surfaces gape open, but when the carcass is hoisted, it is difficult to see into the cut, and a hook is required to pull the two surfaces apart. Several measles were found in an incision about an inch below the pelvic symphysis into the adductors, in carcass No. 23, described above.

In the subjoined observations of various workers, it will be noticed that very few writers in Europe mention incisions into the shoulder-elbow muscles or the thighs. Two notable exceptions are Buri and Krupski, who worked in Switzerland during the Great War period.

There can be little doubt that the recorded incidence of bovine cysticercosis will become considerably higher, if our co-workers in Europe were to incise into, e.g. the *triceps brachii* and the *adductor*

Working on Bali Island, Le Coultre found 1,337 measly carcasses. He divided these into three groups:—

1. *Cysticerci* in head only, in 838 cases.
2. *Cysticerci* in head and carcass, in 300 cases.
3. *Cysticerci* in carcass only, in 199 cases.

If Le Coultre had followed the custom of inspection of Holland and of Germany, he would have missed the 199 measly cases entirely.

It will also be noticed that those writers, who were probably privileged to dissect the entire carcasses, chiefly refer to the muscles of the hind limbs and/or those of the fore limbs as predilection sites. (See Hammer, Teppaz, Valade, Claverie, Alix, Le Coultre, Morot.)

Von Ostertag's expression " the more you look, the more you will find " is very applicable to the meat inspector's search for *Cysticercus bovis*. The inspector's permitted range of incisions is too limited, and although it may be felt that in the standardized routine incisions prescribed in South Africa, he will probably encounter most measles, the possibility of undetected measles in the not incised hind limbs must not be overlooked.

Reference to our table also discloses the frequency with which measles were found in the vertebral muscles and in the extensors and flexors of the carpus.

It is interesting to record that in none of the 25 observed cases did we find measles in any of the viscera (except the heart) nor in any glands. No measles were observed in the fat in these cases, nor in the brain, nor the eyeball.

The hump is very definitely a very common site for measles.

Exceptionally unusual infestations with measles have been encountered in South Africa.

In 1935 we found more than 50 apparently viable measles in the tongue of an ox, and yet we failed to disclose a single measle in any of the secondary incisions prescribed by Regulations. This carcass was not minutely dissected.)

Mr. Thatcher, Health Inspector, Fort Beaufort writes:—

> " I have specimens of measles found in bovines in the kidney, submaxillary and inguinal lymphatic glands, hard fat of the heart and in some instances in other hard and soft fats of cattle."

The Town Clerk, Kimberley, advised me that in one case they found measles in the lungs of an ox.

Mr. W. J. Armstrong, Meat Inspector, Vryheid writes:—

> " I would say the tongue is the commonest site. I had a peculiar experience about a year ago. While the animal was being skinned, I noticed measles lying on the surface side of the shoulder and when I did the shoulder cut nothing was to be found, but found the head, tongue and heart heavily infested."

It has been mentioned that we also studied the predilection sites in 113 consecutive lightly infested carcasses. It was impossible to dissect those carcasses minutely, since, in accordance with paragraph 16 (2) and (3) of Section 115 of Act No. 36 of 1919, as amended by various Government Notices, they were subjected to fourteen days freezing at -10° C. We had, therefore, to accept the number of cysts found in the routine and secondary incisions as indicative of the commonest sites of infection.

Except in one case, in which a viable *cysticercus* was found in the *M. Semitendinosus*, no incisions were made into the hind limb. The measle found in the semitendinosus muscle was encountered purely accidentally. A small piece of this muscle had to be excised from the quarter on account of bruising, and to our utter astonishment we found a viable *Cysticercus bovis* exposed. Not a single measle was found in any of the secondary incisions which were subsequently made. This was another instance in which coincidence fortunately stopped a measly carcass from being passed as fit for human consumption.

In recording the sites of infection in the 113 lightly infested carcasses, we found:—

Measles in the masticatory muscles only, in	40	cases.
Measles in the masticatory muscles plus shoulder, plus tongue in	5	,,
Measles in the masticatory muscles plus shoulder, plus heart in	2	,,
Measles in the masticatory muscles plus shoulder plus psoas in	1	,,
Measles in the masticatory muscles plus shoulder, only, in	35	,,
Measles in the masticatory muscles plus psoas, only in	1	,,
Measles in the masticatory muscles plus diaphragm, plus sternum in	1	,,
Measles in the masticatory muscles plus diaphragm, plus shoulder	1	,,
Measles in the masticatory muscles plus sternum (brisket), only, in	1	,,
Measles in the masticatory muscles plus heart in	2	,,
Measles in the masticatory muscles plus tongue in ...	5	,,
Measles in the shoulder muscles, only, in	10	,,
Measles in the shoulder muscles plus tongue in	5	,,
Measles in the shoulder muscles plus psoas in	1	,,
Measles in the shoulder muscles plus sternum (brisket) in	1	,,
Measles in the tongue plus the heart in	1	,,
Measles in the semitendinosus muscles, only, in	1	,,

Analysing the actual number of measles found, and the various number of cases, we found in the 113 carcasses:—

In the masticatory muscles in	94 cases	129 measles.
In the muscles of the shoulder above elbow	61 ,, ...	113 ,,

In the tongue	16 cases	17 measles.
In the heart	5 ,, ...	6 ,,
In the psoas muscles	3 ,, ...	3 ,,
In the sternal muscles (brisket) ...	3 ,, ...	3 ,,
In the diaphragm	2 ,, ...	2 ,,
In the semitendinosus muscle	1 ,, ...	1 ,,

Thus in 113 lightly infested carcasses we found 274 measles. Of the 129 measles found in the masticatory muscles, 98 were found in the external masticatory muscles and 31 in the internal masticatory muscles.

The ratio of light infestation to heavy infestation is reflected in the following statistics:—

> From July 1st, 1934, till December 31st, 1936, 1,060 bovine carcasses were found to be measly at the Bloemfontein abattoir.

Of this number 953 carcasses were lightly infested, that is, they were treated in the freezing chamber according to Regulations, and 107 were grossly infested, that is more than six measles were found in the incisions into the carcass, excluding the head and the viscera, or a total of ten measles in the carcass, including the head and the viscera.

Mode of Inspection.

Regulations governing the inspection of carcasses for measles were discussed earlier in Part III of this work, in the section dealing with the routine inspection of pig carcasses. For the inspection of bovine carcasses, the routine is the same as that of pig carcasses, except that the following provisions are specifically made:—

> Paragraph 13 (*a*) includes a clause which lays down that the cheek muscles of bovines shall be examined by two or more linear incisions on the outside and a linear incision on the inside, which shall all be made parallel to the lower jaw.

The meat inspector is not authorized to make a routine incision into the substance of the tongue, unless he has found evidence of measles in the routine incisions, nor is he allowed to make any inspection incisions, other than those for the examination of the lymphatic glands, into the pelvic limb.

Various Authors on Predilection Sites and Inspection Technique.

Up till 1888 very little was known of beef measles in Europe, and between the years 1883 and 1889 only four cases of bovine cysticercosis were found at the Berlin abattoir. About that time Hertwig recommended that the muscles of the pharynx should be incised for the examination for rinderpest, and this led to incisions into the internal masticatory muscles. A number of measles was thus found. The method was further improved by Glagé, who incised the external masticatory muscles and found still more measles in those muscles. This led to the first "predilection sites," discovered, as van Oijen (1929) suggests, "by pure accident". Further observations led to the discovery of yet other "predilection sites", for example, the heart, the tongue and later the neck muscles.

Von Ostertag (1913) gives the following table showing the frequency with which beef measles were found between 1888-90 at the Berlin abattoir:—

1. In the masticatory muscles	in	360	cases.
2. In the heart	in	41	cases.
3. In the tongue	in	10	cases.
4. In the thoracic muscles	in	1	case.
5. In the cervical muscles	in	3	cases.
6. In the general musculature	in	22	cases.

He mentions that with the exception of the heart, the vital organs of cattle are not usually infested with *cysticerci*. Only in cases of extensive invasions are the lymphatic glands, lungs, liver and brain infested.

Von Ostertag quotes Morot, who found in an African beef animal that the internal masticatory muscles were less strongly infested than the tongue and the heart. *Cysticerci* were also found in large numbers in the muscles of the *shoulder*, *fore leg*, *back*, *rump* and *hind quarter* (*Cf.* table of infestation in the 25 cases dissected at Bloemfontein.)

Flohil (1910) quotes Beunders, who found at the Groningen (Holland) abattoir the following order of frequency of infection:—

> (1) Heart; (2) Internal masticatory muscles; (3) External masticatory muscles; (4) Tongue; (5) Diaphragm.

Le Coultre, during his investigations in Bali in 1927, had the opportunity to dissect all infected bovine carcasses. Le Coultre had, therefore, the privilege to compile a very accurate estimate of the predilection sites from actual observations of a number of carcasses. At the abattoirs at Boeleleng and at Denpasar the combined number of measly carcasses during 1927 was 937 out of a total of 3,810 slaughtered during that year.

Measles were found:—

In the masticatory muscles	in	778	cases	(83 %)
In the tongue	in	131	,,	(14 %)
In the shoulder muscles	in	114	,,	(12 %)
In the adductors	in	109	,,	(11·6 %)
In the cervicals	in	70	,,	(7·5 %)
In the intercostal muscles	in	45	,,	(4·8 %)
In the psoas muscles	in	42	,,	(4·5 %)
In the infra-vertebral muscles	in	36	,,	(3·8 %)
In the sternal muscles	in	24	,,	(2·

In the diaphragm	in	19 cases	(2·0%)
In the abdominal muscles	in	17 „	(1·8 %)
In the heart	in	14 „	(1·5 %)

He also found 5 cases with measles in the oesophagus, 4 in the hump, 1 each below the elbow and the patella, 1 in the brain and 1 in the kidney.

Alix (1887), working in Tunis, found the following order of frequency of infection :—Tongue, heart, muscles of the thigh, muscles of the shoulder, croup, intercostals, pectorals and psoas.

Hammer wrote in 1922 that in former German East Africa he found the most important predilection sites to be :—The adductors, the muscles of the neck, the tongue and lastly the heart. Hammer was privileged to incise other parts of the carcass than those prescribed in customary European inspection technique. According to Capt. H. J. Lowe, M.R.C.V.S., of the Department of Veterinary Science and Animal Husbandry, Tanganyika, who wrote to me in 1936, the chief predilection sites in Tanganyika at present are—(1) the heart; (2) the muscular mass of the upper part of the hind leg, i.e. Biceps femoris and Semitendinosus and also the Triceps of the arm; (3) the tongue; (4) the masseters.

Teppaz (1923) working in Dakar (Senegal) found the following order of frequency :—Cervical muscles, masticatory muscles, heart, diaphragm and adductors.

Vallade (1927) gave the following order of frequency as representative of his observations at Homs (Syria).—Diaphragm, heart, psoas muscles, masseters, adductors and cervicals.

Claverie (1928) found the anconeus muscle the most frequent site of infestation in French Guinea. It is difficult to understand how he incised the anconei and found more measles there, than in the larger superlying triceps muscles, in which group measles are, definitely, very frequently found. Claverie's custom was to make two incisions into the anconeus muscle on each side.

Prof. S. Yoshida of the Osaka Imperial University, Osaka, Japan, supplied a translation of a recent paper written by Nakanishi, who found the following order of infestation in Korea: Heart muscle 75 per cent. of cases; trunk muscles 47·7 per cent. of cases; tongue 30·0 per cent. of cases; masticatory muscles 23·5 per cent. of cases; diaphragm 20·3 per cent. of cases; lungs 19·77 per cent. of cases; cutaneous muscles 18·9 per cent. of cases; retina 10·5 per cent. of cases; pericardial sac 9·1 per cent. of cases; gastric wall 8·5 per cent. of cases; lymph glands 6·5 per cent. of cases; oesophagus 5·2 per cent. of cases; kidneys 5·2 per cent. of cases; pancreas 3·9 per cent. of cases; bladder 3·3 per cent. of cases.

Nakanishi, therefore, gives a totally different order of infestation, and he found the masticatory muscles to be, relatively, of much less importance as a predilecton site, than most European writers. The comparatively high percentage of cases with pulmonary, retinal and lymph glandular cysticercosis is also worthy of note.

Ransom (1911) described the predilection sites observed in the United States as Heart 70 per cent., masticatory muscles 47 per cent. of cases. Dr. Mohler, Chief of the Bureau of Animal Industry, United States Department of Agriculture, informed me that the most prevalent seats of infection in the United States at the present time are: (1) The muscles of mastication. (2) The heart. (3) The muscular portion of the diaphragm.

Veenstra (1921) found the following order of frequency at Amsterdam: Out of 26 single measled cattle he found the external masticatory muscles infested 16 times, the heart 5 times, the tongue 3 times, the internal masticatory muscles twice.

Reitsma (1931) found at Rheeden (Holland) the following order of frequency: (1) Heart. (2) Left external masticatory muscles. (3) Right external masticatory muscles. ((4) Diaphragm. (5) Left internal masticatory muscles. (6) Right internal masticatory muscles.

Reitsma mentions an interesting fact, to which he cannot ascribe any reason, namely that he, and some other observers have found more measles on the left side (masticatory muscles) than on the right side. Most authorities maintain that the majority of measles are found near the edge of the jaw bone, but Reitsma and Veenstra disagree with this view.

Van der Slooten (1936) wrote that it was doubtful whether the so-called predilection sites were truly the most common sites of infestation. He found that the hump was commonly as heavily infested as the usual muscle groups inspected. (Note that at Bloemfontein we found 98 measles in the hump and cervical muscles in 20 out of 25 grossly infested carcasses.)

Buri (1915) and Krupski (1917) found the following order ot infestation at Berne and Liestal, respectively:—

Both observers: Masticatory muscles, heart and diaphragm.

Buri: Rarely in the tongue, sternal muscles, biceps femoris and intercostals.

Krupski: Rarely in the tongue, shoulder muscles, abdominal muscles and gracilis muscle.

Funck (1930) pointed out that next to the masticatory muscles the oesophagus is the most common site of infestation. During four years' observation at Neumunster, out of 120 adult bovines with live *cysticerci*, he found: 105 cases of measles in the masticatory muscles, 13 cases in the oesophagus, 5 cases in the heart, 5 cases in the abdominal muscles and one case in the diaphragm. *Cysticerci* thus appeared in the oesophagus in 10·3 per cent of cases, and in all these 13 cases only one live measle was found in the whole carcass. In 1935, Funck recorded that in five cases he found measles to be extremely shallow in various facial muscles, the lips, etc., and not necessarily in the usual sites. Funck advised that these superficial muscles should receive careful attention. He also maintained that all ox heads should be examined on a table, so that the light could be shone more readily into the cheek cuts.

Cattoneo (1932) also found that the oesophagus could be considered an important predilection site of *C. bovis*. Out of 40 cases investigated, he found measles in the oesophagus in 17 cases.

Messner (1931) described a case of a 6-years-old ox in which a number of *cysticerci* was found in the oesophagus only, in Karlsbad.

Coussi (1933) found at the abattoirs at Sousse (Tunis) that the heart was the most common site to be infested, during five years' close examination. Out of 621 animals infected 524, i.e., 84·37 per cent. showed cardiac *cysticerci*; 318, i.e., 51·2 per cent. showed measles in the masseters; and 260, i.e., 42·19 per cent. in the tongue.

Krueger (1935) found the predilection sites to be, in order of frequency: Masseters, tongue, diaphragm and heart. The heart was infested in only 10 per cent. of cases.

Stengel (1932) advocated opening the pericardium in each carcass, since he frequently found measles just below the pericardium.

The necessity for careful inspection of the liver for *cysticerci* has recently been mentioned by Poisson (1934), by Buck, Lamberton and Randriambeloma (1935) and previously by Schlegel (1918). Poisson records a case of infection with *Taenia saginata* in a patient in Madagascar, as the result of the ingestion of a prescribed raw liver diet. Buck and his co-workers found two *C. bovis* in the liver of a cow. Schlegel found a *C. bovis* in the liver of a cow. It was the only measle found in the carcass.

Professor P. G. Malkani supplied me with photographs of *cysticerci* in the heart, liver and lung of a bovine in India. (See Fig. 3.)

Mahlendorff (1929) names a case in which he found a *C. bovis* in the subcutaneous fascia and in one kidney, while the usual predilection sites were quite free.

REMARKS ON INSPECTION TECHNIQUE AND RECOMMENDATIONS BY SOME WRITERS.

Many recent workers in Europe have stressed the point that meat inspectors should be allowed greater authority and more liberty for inspection. For example, in 1932 B. Müller suggested the standardization of inspection technique in Germany, by making two incisions into each masseter. The masseters are considered the most probable site of infection by workers in Europe, and many consider that by increasing the number of masseteric incisions, a very much larger percentage of measles will be found, and that there will be a corresponding impetus towards the eventual eradication of bovine cysticercosis. Among these writers mention may be made of Künibert Müller, quoted by Le Coultre, who in 1905 recommended that the external masticatory muscles should be incised onto the *crysta zygomatica*, and the flaps formed turned right back. By this means Müller found 4·6 per cent. infected at Guben, and Junack 2 per cent. in Kottbus.

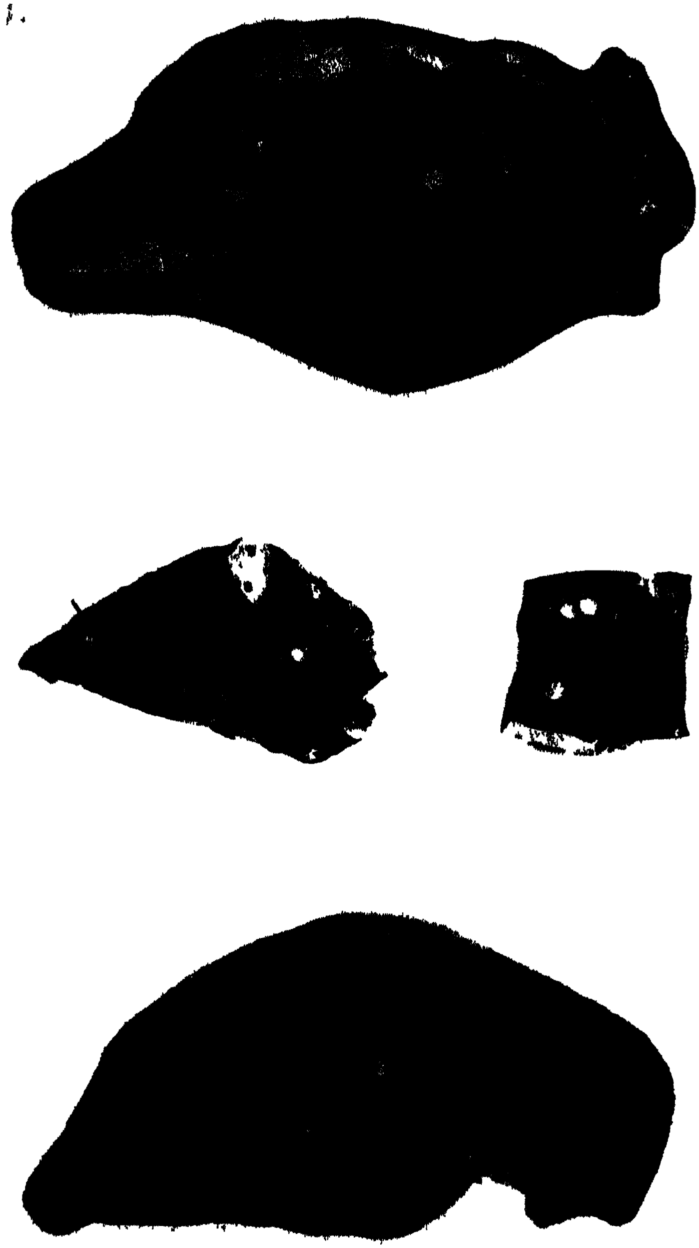

Fig. 3.—Appendix "A" Photographs of *Cysticerci bovis* of the heart, liver and lung of an ox. Reproductions of photographs supplied by Prof. P. G. Malkani, Patna, India.

K. Müller (1927) recommended doubling the number of incisions into the masticatory muscles. He also advised making incisions into the muscles under the tongue. All incisions should be examined very carefully, and only sharp, firm knives should be used. To ensure efficiency, not more than 50 to 60 bovines should be inspected by one inspector per day.

Mahlendorff (1930) found 0·81 per cent. to 0·91 per cent. measles in cases where one cut was made, and 1·06 per cent. to 1·54 per cent. in cases where more than one cut was made. Mahlendorff mentioned that prior to June, 1929, only one incision was made into each masseter at Breslau. In April, 1929, he found 0·81 % infected, and in May 0·94 per cent. In June that year he made two incisions into each masseter and found in: June 1·11 per cent.; July 1·39 per cent.; August 1·06 per cent; September 1·11 per cent.; October 1·53 per cent.; November 1·54 per cent.; December 1·22 per cent.; and in 1930 in January 1·42 per cent.; February 1·35 per cent.; March 1·08 per cent.

Kern (1930) expressed the opinion that the problem of eradication of *Taenia saginata* could be solved by thorough inspection technique. He always insisted on two incisions into the external masticatory muscles, and if possible into the internal masticatory muscles as well.

In *Zeitschr. f. Fl.-und Milchhyg.* 40, p. 386, von Ostertag (1930) recommends a double incision of the masticatory muscles, and if necessary, even transverse cuts, when many more measles will be found.

Platschek (1931) recommended three or even four incisions into each masseter. Similar recommendations were made that year by Wernery, who advised that the number of incisions in the external masticatory muscles be increased from two to three on each side.

The new inspection technique in Germany, since 13th October, 1934, provides, according to personal information supplied by Dr. Heinrich Wagemann: " A careful inspection of the tongue, heart and external and internal masticatory muscles. At least two incisions must be made parallel to the mandible, and while the carcass is being dressed, cut surfaces must be inspected for measles. The incisions into the masticatory muscles must be made from the border of the mandible to the upper half of the inside of the jaw, and as far as it can be cut into, upwards to the lymphatic glands of the ear on the outside of the jaw."

All German writers, however, do not necessarily hope that a greater number of incisions into the masseters will be the main solution of the taeniasis problem. Several writers stress the fact that the low incidence recorded at certain abattoirs is mainly due to slackness in inspection technique. Among these Junack was a leader. Junack (1926) formed the following conclusions: " The apparent big decrease in the incidence of cysticercosis in some areas may be attributed to careless inspection during and after the war, owing to shortage of staff, or owing to various modifications in inspection, or owing to removal of *cysticerci*, surreptitiously, by butchers themselves. Other local difficulties may also be responsible." In support of his statements, Junack quotes:—

"The incidence of measles in Berlin was 22 times as high as that of the outlying areas (*Auszenbezirke*), whereas actually the slaughter stock came from the same regions." Furthermore, according to Junack, in 1923 and 1924, 101 measly cattle were found at Bremen, whereas not a single case was found at Mecklenburg Strelitz, Schaumburg Lippe and Hohenzollern. In 1931 Junack wrote that during the war meat inspection was not too thorough, and it was possible that in some places where troops served it might have been absent altogether. Under such conditions soldiers acquired tapeworm infection and later, in turn infected German cattle with *C. bovis*. Owing to closer meat inspection more cases of *C. bovis* have been found, but in Junack's opinion, yearly 5,000 to 6,000 more cases may be found, if inspection technique were still more thorough.

Similar views were expressed by Profè (1934), who pointed out that it was difficult to compare the incidence of *C. bovis* in various places, because there was a big variance in the thoroughness of meat inspection.

In addition to advising uniformity in inspection technique, Zeug (1931) mentioned that the number of incisions required, and their locations, should be definitely stipulated in Regulations. That the human element was an important factor in the discovery of *cysticerci* was clearly shown by Zeug. Thus, there was a big variation in the efficiency of the work performed, comparatively, in the following groups of inspectors:—

(1) The number or percentage of full-time abattoir veterinarians who found measles.

(2) The number or percentage of veterinary practitioners, who spent a small part of their time at abattoirs, and who found measles.

(3) Unqualified assistant inspectors, who found measles.

Zeug qualified his remarks by giving actual statistics.

Wernery (1931) blamed perfunctory meat inspection for the failure to eradicate bovine cysticercosis. He mentioned several instances in which a much higher percentage measles was found after deeper and larger incisions of the masticatory muscles were made.

After having made only one incision into each masseter, Juraske found only 0·16 per cent. of cases measly at the Jena abattoir, but when he increased his masseteric incisions to two on each side, Juraske found that the percentage had increased to 1·29. (Wernery, 1931; von Ostertag, 1930.)

Wernery recommended that the Regulations should lay down precisely, as to where and how incisions should be made, and mentioned that in certain parts of Prussia, where specific incisions were made, the percentage measles was much higher than in other districts.

Buri, working in Switzerland in 1914 and 1915, caused a big increase in the number of *C. bovis* carcasses to be found at the abattoir at Berne, where he incised the masticatory muscles freely. The result of his system of incisions was an increase in the number of cases from 1 and 5 in 1912 and 1913, respectively, to 23 and 39 in 1914 and 1915, respectively. Buri was convinced that nine-tenths of the cases of *C. bovis* found, resulted from the extra incisions into the masticatory muscles, and he persisted with this practice, despite the determined protests of the butchers. It was he, who in 1915 recommended that Switzerland should adopt the German method of inspection, since the incidence of *C. bovis* was high and warranted it

Guillebeau (1917) objected to the severe German mode of inspection, and considered that this method was too rigid for Switzerland, in which country raw, or insufficiently cooked meat was seldom eaten, although he freely admitted that the incidence of taeniasis was high in those parts of Switzerland, where the so-called *Landjäger* was commonly eaten.

The same year, 1917, Krupski attacked the mild views expressed by Guillebeau, and proved that at Liestal he found a percentage of 5·9 in cases in which he freely incised the masticatory muscles. He was a staunch advocate for the introduction of the German method of inspection, into Switzerland. Krupski compared the great variation in inspection technique and mode of control of cysticercosis, as was practised at various Swiss centres. Thus he quotes:—

Zürich—only the predilection sites which could be inspected without incisions, and thus not the masticatory muscles, were inspected.

Basel—*idem*, and two masticatory muscles were incised.

Schaffhausen—*idem*, and besides an attempt was made to detect the responsible tapeworm carrier by investigations at the place of origin of the infected bovine.

St. Gallen—*idem*.

The American Edition (1934) of the work by Edelmann, Mohler and Eichhorn states that it is absolutely necessary to make several cuts into the inner and outer muscles of mastication, to inspect carefully the tongue and its musculature, and also to inspect carefully the heart, externally and internally, after laying open the chambers and cutting through the dividing wall. It is understood that all surfaces as well as cut surfaces of the remaining muscle should be inspected for beef measles.

Leighton (1927) quotes that the following inspection custom is followed in the United States:—

> "The presence in the flesh of cattle of a certain cyst capable of producing tapeworm in man can usually be detected by examining the inner and outer cheek muscles. Therefore, these muscles are laid open by slicing-cuts for the detection of the cyst."

The United States of America Bureau of Animal Industry Order 211, Regulation 11, Section 16, Paragraph 3 defines a careful examination of the heart, muscles of mastication, tongue, diaphragm and its pillars and those portions of the carcass rendered visible by the dressing.

In Canada, although the incidence of bovine cysticercosis is relatively low, it would appear that a thorough inspection of the head is made, similar to the practice formerly in vogue in Germany and in Holland. Paragraph 12 of the Canadian Meat Inspection regulations reads:—" The tongue must be so loosened as to expose the internal muscles of mastication. These, and the external muscles must be incised, cutting parallel with the lateral surface of the jawbone, the cut surfaces to be minutely examined. The surface of the heart must be closely scrutinized and the heart then opened or inverted. This can best be done by placing the left ventricle uppermost, when one incison, the full length of the organ, will be sufficient to permit an examination of the cut surfaces and of the interior; or the heart may be everted and incisions made into the musculature of the organ."

Working in Denmark, Nielsen (1934), showed that on several occasions infected carcasses could have escaped detection owing to the absence of *cysticerci* in the recognised predilection sites, or owing to a perfunctory inspection.

As regards inspection technique in Holland, Tenhaeff (1907) wrote that in Utrecht up till that year only the internal masticatory muscles were incised. Up till 1905 approximately only two measly cattle per annum were found at that abattoir. In 1907 von Ostertag visited the Utrecht abattoir, and pointed out the desirability of incising the external masticatory muscles as well. Coincidentally, von Ostertag demonstrated the method of incision into the external masticatory muscles and found a degenerated *cysticercus* in his incision. That season Tenhaeff found 74 cases of cysticercosis, after having followed von Ostertag's recommendations.

Veenstra (1921) laid special stress on the careful inspection of the masticatory muscles. He recommended two large incisions, and incisions into the tongue. If necessary, the masticatory muscles should be cut into thin strips. He felt convinced that only the inspection of the predilection sites would be sufficient to eradicate taeniasis-cysticercosis in a decade. Veenstra recommended the following technique:—

Masticatory muscles.—Two parallel incisions through each external masticatory muscle, and one deep incison into each internal masticatory muscle.

Heart.—First external inspection, then an incision into the left ventricle up to the septum; sub-division of both halves of the left ventricle into flakes like an onion; then a longitudinal incision through the right ventricle. The cut surfaces and the endocardium to be thoroughly inspected.

Tongue.—Thorough palpation and incision of the muscles.

Diaphragm.—Inspection and palpation. (No mention of incisions.)

Reitsma (1931) quotes Article 31 of Section 285 of the Netherlands Meat Inspection Regulations of 5th June, 1920, which laid down that the tongue, heart and external masticatory muscles were to be incised—the latter by " various longitudinal cuts ". Reitsma strongly recommends that five to ten transverse incisions be made as well. By doing this he claims a far more thorough inspection, as was proved by his results.

Schoon (1933), in his discussion on the eradication of bovine cysticercosis, based on his experience in meat inspection, mentions that the high incidence of *C. bovis* (4 per cent.) observed at Nijmegen, is due to his method of inspection. Two even, clear cuts are made into the masticatory muscles on either side of the jaw, and three into the heart. Schoon mentions that the large percentage of cases with a single measle is notable.

Professor C. F. van Oijen of Utrecht, Holland, informed me (1936) that at Rheeden in 1934 special attention was directed towards the muscular coat (*spier-rok*) of the oesophagus. The mucous membrane (*slijmvlies*) was excised, stretched out and carefully inspected. This technique was responsible for the finding of thirteen measles, of which three appeared to be viable. According to Prof. van Oijen, the increased incidence of measles in some parts of Holland can mainly be ascribed to the result of more thorough inspection technique.

At Karlsbad (Bohemia), Messner also paid special attention to the inspection of the oesophagus.

In Great Britain, it must be freely confessed, inspection technique for *Cysticercus bovis* falls far short of that practised in Holland, Germany, Denmark, or particularly in South Africa.

In Scotland, Regulations prescribe that " the cheek muscles shall be examined by a linear incision parallel to the lower jaw." Gerald Leighton (1924), writes:—" A special instruction states that an examination must be made of the cheek muscles, and this must be done by cutting into them in a line parallel to the lower jaw. This is a proceeding which has not been hitherto very common in this country, though a few inspectors carry it out. It is, however, a very important point, and the object is to ascertain whether the muscle is infected with a parasite known as the ' beef bladderworm ' or ' beef measles '." Leighton's quotation is not from the lay press, or from a non-technical article to farmers, butchers, or other laymen interested in the meat industry, but actually appears in " A Handbook of Meat Inspection—A guide to the Public Health (Meat) Regulations (Scotland) 1924 ". Leighton, therefore, frankly informs students and others interested in the science of meat inspection that up to 1924, although regulations provided for a cursory inspection for measles, little or no notice was taken of them.

The present writer is probably not alone in the surmisal that if a mode of inspection on the German principle, which is not as complete as that of South Africa, be instituted into Great Britain, the incidence of measles in that country will be considerably higher, and may even startle some authorities.

Colonel T. Dunlop Young, one of Britain's greatest authorities in meat inspection, kindly agreed to enquire into the incidence of measles in Britain, from records of most of the principal abattoirs. On my behalf, he very kindly instituted a searching enquiry, and he failed to find a record of a single instance of *cysticercus* infection in the reports of the various abattoirs during 1935. It seems almost incredible that the incidence of *C. bovis* could have been " nil " in Great Britain.

In 1920, Robertson sounded a warning to meat inspectors and meat consumers in Great Britain. In his " Meat and Food Inspection ", pp. 136-138, he states that inspection of meat for measles in Great Britain should receive greater attention. The possibility of infection of his patients with *T. saginata* was forcibly brought home to him when, as Medical Officer of Health for Leith (now a suburb of Edinburgh), he prescribed a raw meat diet for tuberculosis cases at the Leith Isolation Hospital. Several of his patients developed tapeworms.

For Syria, Valade (1927) recommended the following inspection:

(1) Excission of the diaphragm, which must be hung on hooks for careful examination.

(2) Heart: Careful inspection of the coronary grooves. Then an incision into, and inspection of the myocardium and the endocardium.

(3) Psoas muscles: These must be examined along their entirety. (In Vallade's opinion *cysticerci* are more frequently found superficially in the muscle fascia than in the muscle fibres.)

(4) The external masticatory muscles must be cut into thin strips. (In the event of one or more measles being found in the above four sites, then the following incisions should be made.)

(5) A series of incisions must be made plumb down the medial surface of the thigh into the adductor muscle.

(6) Incisions into the superficial cervical muscles.

In Bloemfontein we cause two long incisions to be made, widely apart and parallel to the lower border of the mandible, and to pass in an upward direction to sever the parotid gland. This procedure has been in practice since the middle of June, 1934, when my Senior Meat Inspector, Mr. H. M. Downes, and I assumed office shortly after each other at this abattoir. The internal masticatory muscles are cut with two incisions on each side, as long as possible. The effective series of incisions into the masticatory muscles gave the following immediate results.

June, 1934	39 cases (between 18th June and 30th June).
July	64 cases.
August	65 cases.
September	57 cases.

Mr. Downes assumed duty on June 18th and prior to his arrival, namely from 1st to 17th June, 1934, only 7 cases of measles were found. During May, 1934, 13 cases were found; during April, 1934, 15 cases were found.

These figures prove that effective incisions and efficient work is rewarded with successful results. The slaughter stock belonged to the same firms of butchers throughout the period recorded, and originated from the same parts.

We were blamed for the butchers' misfortunes, but, despite the complaints from the butchers that we were too drastic with our technique, I insisted on the two long incisions into the masseters, and instructed Mr. Downes and the Junior Inspector to persevere with the practice. It is is also interesting to relate that the Superintendent of one of our larger Union abattoirs visited Bloemfontein some time ago, and, upon witnessing our technique, remarked: " If we were to make those cuts in our abattoir the butchers would revolt! "

The writer has, indeed, noticed at some of our larger abattoirs, and at nearly all the smaller abattoirs which he has visited, that the masseteric incisions, and frequently those into the triceps brachii are far too short and shallow to be effective.

Our South African meat inspectors should bear in mind that Regulations restrict their routine incisions to a bare minimum, and in those parts of the carcass where they are expected to make their incisions, and the size of such incisions is not definitely prescribed, they should err on the side of long and deep cuts and should determine to make these as long as possible.

THE AGE AND SEX OF INFESTED ANIMALS (EXCLUDING CALVES).

It has already been mentioned that during the period 1st July, 1934, to 31st December, 1936, 1,060 cattle were found to be measly at Bloemfontein abattoir. The total number of bovines (excluding calves) slaughtered was 21,764, giving a percentage of 4·87.

We noticed that there was very little difference in the percentages measles among cows and oxen, but bulls were relatively less frequently infested.

The following table shows the numbers and percentages, also the ratios of light to heavy infestation in the sexes.

	Total stock slaughtered.	Found measly.			Percentage.
		Total.	Heavily infested.	Lightly infested.	
Oxen...........	19,305	957	94	863	4·96
Cows...........	2.249	99	12	87	4·40
Bulls...........	210	4	1	3	1·90
TOTAL......	21.764	1,060	107	953	4·87

Several writers in Europe mention the disproportion of infestation between the various sexes.

Krueger (1935) found that the incidence in bulls was twice that in cows.

Braun-Seifert (1923) quotes an extract from "*Ergebnisse der Schlachtvieh -und Fleischbeschau im Deutschen Reich*" for 1905, in which the incidence of bovine measles was analysed as follows:—Bulls, 0·6 per cent.; oxen, 0·56 per cent.; cows, 0·17 per cent.

Von Ostertag explains the comparatively heavier incidence of cysticercosis in the male sex by the fact that male bovines are generally slaughtered early, whereas infection is usually acquired early, and that the bladderworms die off later, at about the usual age that cows are slaughtered.

In Bloemfontein, in 215 consecutive measly carcasses, we found—

74 carcasses to be over 5 years old;
126 carcasses to be 4 to 5 years old;
15 carcasses to be 3 years old.

Admittedly, these figures are no criterion, since the numbers of animals slaughtered at the various ages were not included in our recordings. The vast majority of cattle slaughtered at this, and indeed at most South African abattoirs are well over three years old.

Flohil (1910) mentioned that Beunders at Groningen (Holland) found a percentage of 0·41 infected between the years 1904 and 1908. According to Beunders, cysticercosis was equally found in cases under two years old and in cases above that age.

DEGENERATION OF THE CYSTICERCUS BOVIS.

Degeneration of the beef bladderworm occurs readily, and according to some authorities fairly early. (*Vide* frequency of degenerated *cysticerci* in calves.) Cysts in the various stages of degeneration are very frequently encountered on meat inspection.

In Germany and in Holland recorders frequently discriminate between degenerated and apparently viable cysticerci. Judging by various reports, it would appear that many of our co-workers in Europe make a distinction in their returns between dead and live measles, although all carcasses showing measles, whether dead or alive, may be treated in the same way.

Apparently the Regulations in Germany are similar to those of South Africa, in so far as that all carcasses are condemned or detained for treatment (e.g. by freezing), irrespective of whether viable or degenerated *cysticerci* are found.

Hock (1934) criticises the German custom. He considers it quite unnecessary to condemn carcasses containing only dead *cysticerci*. If the cysticerci are so numerous as to render the meat "substantially changed", then it can be condemned under other sections of the Regulations. If the dead *cysticerci* are few, then the meat is, in Hock's opinion, not unfit for human consumption.

Most observers have argued against the views expressed by Hock. The possibility that dead *cysticerci* and live bladderworms may be present in various parts of the same carcass, has been recorded by several writers on the subject.

Prillwitz (1930) pointed out the fact that although only degenerated measles may be found in a certain part of an ox carcass, it may be possible that live measles may be present elsewhere in that carcass, owing to the fact that the animal may have become reinfected owing to its proximity to a tapeworm carrier. He quotes two cases in which he found only one totally calcified measle in the masticatory muscle, and with further inspection, he found viable measles in the tongue muscles.

Haas (1929) found numerous measles in a very young calf. These were diagnosed at the Veterinary School at Albert to consist of both degenerated and viable specimens.

Holtz (1929) recorded two cases in calves. In the first case eight calcified and three viable measles were found in the heart, and a number of dead measles and two living measles in the masseters. In the second case Holtz found two viable and six dead measles in the heart and three measles in the external masticatory muscles, and also three live measles in the diaphragm. Further examination produced a number of translucent cysts with apparently healthy scolices in the superficial fascia, hump, shoulder, hind quarters, etc.

Le Coultre (1928) mentioned several cases in which he found both living and degenerated *cysticerci* in the same carcass.

Reitsma (1931) recorded finding three living measles in the heart, after having found many dead measles elsewhere.

Van der Slooten (1936), writes:—" It is of little importance as to whether the parasites found are alive. We must bear in mind the fact that even though a parasite may be dead, others, elsewhere in the carcass may still be alive. There is no reason to pass as fit for human consumption meat which may merely show one or two degenerated measles." He continued:—" It is important to examine any groups of calcified measles very carefully, since one may frequently find a perfectly normal and viable *cysticercus* in the area covered by the calcified cysts."

It is, therefore, right to assume that any cases showing only degenerated measles in the standard routine incisions, may, nevertheless, have some living *cysticerci* elsewhere in the carcass. For that reason we made no attempt at the Bloemfontein abattoir to record separately the actual number of cases showing degenerated cysts and the number showing only apparently viable cysts. I agree with the views expressed by Van der Slooten that it is of no importance as to whether cysts found are dead or viable.

In the 25 heavily infested carcasses which were closely studied, the measles were all viable. Those carcasses were specially used for viability tests after various periods of freezing, and superficial measles were, in each case, tested by Keller's simulating infection test for viability, before the carcasses were frozen. It is a remarkable fact that from 1st May to 31st December, 1936, only two carcasses, which were totally condemned, were not so used, owing to only degenerated measles having been found in the inspection incisions.

During 1936, however, my attention was drawn to four measly carcasses in which we found both degenerated and apparently viable *cysticerci*. These were the only four cases which we observed, but there were probably several more.

The process of degeneration, namely the progressive stages of caseation and calcification, is similar to that of *C. cellulosae*. Caseation follows the death of the bladderworm, and is succeeded by the deposit of calcium salts first in the outer capsule and later in the vesicle itself. It has not yet been established how long, under normal circumstances, a mature *cysticercus* will live, before death and progressive degeneration will result. It has been found that *cysticerci* may live in the ox for periods in excess of one year.

In an experimental calf killed 244 days after experimental feeding, Saint-Cyr found only dead cysticerci, the majority of which were in an advanced stage of calcification (Neumann). Simmonds and Cobbold, saw numerous yellow points—chalky deposits, which were dead and calcified *cysticerci* in the muscles of a heifer killed more than a year after the first experimental feeding (Neumann).

Clarenburg (1932) describes his finding a few degenerated *Cysticerci bovis* among numerous (40) living *C. bovis* in an experimental calf killed exactly nine months after the first feeding.

Degenerated or even calcified cysts may be found in the same host with young, viable *cysticerci*, owing to the fact that the particular bovine host may, over various periods of time, have acquired two or more separate infections with *T. saginata* ova.

Daubney (1926) records an interesting fact that has recently been discovered by research, namely that calcification of worm cysts may be greatly accelerated by a course of treatment with the calcifying vitamin, vitamin D, which is present in codliver oil and other fish oils. It is necessary to administer the oil every alternate day for a period of a few weeks, and one must give an overdose, which does not leave a great margin of safety.

This method, mentioned by Daubney, may be of academic and scientific interest, but it will not be of much use in practice.

1. Measles cannot be diagnosed clinically.
2. Serological tests for measles are equally unpractical and non-specific.
3. It would be quite senseless and extremely expensive to treat herds of cattle from areas where the incidence of measles may be high, over a prolonged course. There will only be an effect on the small percentage of measly bovines, which may then be assisted towards more rapid calcification of those measles, and the calcified measles may or may not, later, be detected at the abattoir.

Very interesting research on the subject of immunity of cattle to *Cysticercus bovis* was recently done by Penfold, Penfold and Phillips in Australia. These authors describe their findings in the *Medical Journal of Australia* 1 (13) pp. 417-423 (1936). They refer to a survey by Clapham (1933) on immunity to helminths, and if her survey is complete " only one instance of immunity to the larval stage of cestodes has been proved." The writers and Clapham refer to the work of Miller and Massie (1932), who have shown that the albino rat can be immunized against *Cysticercus fasciolaris*, the larval stage of *Taenia taeniaeformis of* the cat. The writers also refer to immunity to adult cestodes, as worked on by Turner, Berberian and Dennis (1932-33), which work has " probably great practical possibilities in preventing hydatid in man and other animals."

Penfold and his co-workers artificially infested 88 oxen with *Taenia saginata*. These oxen all developed *C. bovis*, that is none were immune. The workers therefore assumed that thirty oxen, acquired from the same parts were also free from infection at the time of commencement of their experiments, since the incidence of infection in Victoria was very low, other than in cattle which had been grazed on a sewage farm. These thirty cattle were drenched with 400,000 *Taenia saginata* eggs (carefully counted), and Penfold and co-workers estimated that 11,000 to 30,000 *cysticerci* developed in each ox. In this way they studied the rate of degeneration, and absorption of the measles. In these heavily infested oxen no live *cysticerci* were found that were older than nine months and *cysticerci* older than seven months were seldom found. " Almost all cysts ten months old or more had contents which were dry dirty yellow and hard, but they were never so hard that they could not be crumbled between the finger and the thumb. The young degenerated cysts of recent infestations had moist green pasty contents."

Penfold and co-workers then drenched three oxen with 400,000 viable *Taenia saginata* ova. A fourth ox from the same batch was not drenched and the four (three artificially infested and one not infested) were depastured on non-contaminated land for fifty-three weeks and five days. After that time, i.e., 30.1.1935 two of the already infected (drenched) oxen, and the undrenched ox were given a drench containing 400,000 *Taenia saginata* ova. All four oxen were slaughtered on 17.4.1935, that is eleven weeks after the second drenching (30.1.35), or sixty-five weeks after the first drenching. The originally undrenched ox showed definite evidence of a recently acquired infestation, that is, as the result of the drenching on 30.1.35. Only about one in every hundred measles were still alive, which the authors said " was quite consistent with an infestation of only eleven weeks of age."

The ox which was not drenched the second time, that is on 30.1.35, showed a recovery of the primary infestation of sixty-five weeks' duration. Only two dead *cysticerci* were found in the whole carcass, and these were approximately one millimetre in the widest diameter. The other two oxen, namely those which had undergone two drenchings at fifty-four weeks' intervals were found to have almost recovered from the original infestation, and immune to the second infestation, i.e. after sixty-five weeks. Experiments were then similarly repeated to determine whether immunity still remained seventy weeks after the cattle were artificially infested. They found the same results, and concluded that "at least some oxen, seventy weeks after being heavily infested with *Cysticercus bovis*, are immune to further infestation; two oxen showed no significant signs of a very heavy primary infestation of seventy-nine weeks duration."

In discussing the practical application of their immunity tests, Penfold, Penfold and Phillips state: "If a live vaccine were to be used and the cattle given the disease, it would be advisable to determine the following: (i) the minimum dose of eggs required to produce a solid immunity; (ii) the stages at which the immunity develops and when it disappears, if at all; (iii) the age at which all cysts die when cattle are given the minimum immunizing dose; (iv) the time necessary for all cysts to be absorbed in cattle immunized with the minimum immunizing dose."

The authors suggest that immunity probably shows itself in two ways. First, as it develops as a result of the primary infestation it kills these primary immunizing *cysticerci*. Secondly, having developed, it prevents the eggs subsequently ingested from developing into *cysticerci*. As the immunity is probably quantitative, cysts may possibly take longer to die and, therefore, to be absorbed, if only a few are present.

SYMPTOMS AND DIAGNOSIS OF CYSTICERCOSIS BOVIS.

Clinical symptoms of cysticercosis in bovines are even more rare than in porcine cysticercosis. Manual examination of the tongue has almost invariably led to negative results. Neumann, however, quotes J. Fleming, who stated that the *cysticerci* may be recognised by examining the tongue, on the lower surface and sides of which they form more or less salient projections, which roll under the finger when pressed upon. Fleming went further and stated that he found on the side of a tongue the largest *cysticercus* he ever encountered, nearly 4 cm. long! It is extremely doubtful if Fleming was actually dealing with *C. bovis*.

After artificial infection, when large numbers of proglottides and ova have been fed to the subject, clinical symptoms may, however, appear. The severe results on the host, in Leuckart's artificial infections of calves were mentioned in Part I of this work. Most of the workers who confirmed Leuckart's experiments, observed clinical symptoms in their subjects. Masse and Pourquier noticed that their experimental calf became greatly emaciated, after showing signs of illness. Zurn's calf showed a temperature of 40° C., rapid pulse, tympanites, emaciation and difficulty in rising. After the calf died Zurn found that infestation was generalised, but the heart was particularly heavily infested. (Neumann.)

Hutyra and Marek mention that Ciga noticed severe cysticercal lameness in an ox. Schmidt found a cyst in the anterior chamber of the eye of a bovine. Ottele noticed high temperature, rapid pulse, laboured breathing and intense itching of the head in a 10 years old cow, as the result of *cysticerci*

Zwijnenberg (1920) recorded a case in a cow, which showed the following clinical symptoms. The temperature was 41° C., frequent pulse, irregular and hardly perceptible. Appetite was diminished, peristalsis normal in quantity, but slightly intensive; rumination was irregular and totally absent from time to time; faeces normal in consistency; salivation light. Milk was withdrawn. At first he suspected foot and mouth disease, which existed in the neighbourhood. After a few days he ruled foot and mouth disease out of the question, but diagnosed septic myocarditis, on account of the cardiac symptoms. At the request of the owner he, nevertheless, treated the animal on rational lines, without success. After four more days he noticed further complications, e.g. photophobia, severe lachrymation, hypopyon. Eventually the owner agreed to the destruction of the cow. On autopsy Zwijnenberg noticed that the large muscle groups were " sowed " with gray cysts the size of peas. These were also found in the masticatory muscles, the heart, lungs, kidneys, salivary glands and the udder. In the myocardium alone, Zwijnenberg found some sixty cysticerci. Microscopical examination proved definitely the diagnosis of *Cysticerci bovis*.

An interesting case of cerebral *Cysticercus bovis*, complicated with generalized tuberculosis was related by Hoefnagel (1923). He stated early in 1923 a bovine from the district was brought to the Utrecht (Holland) abattoir. Before slaughter it was noticed that the beast had an " unsteady " gait. Furthermore, the animal persisted in moving forwards with the head high. After slaughter it was seen that the bovine had a generalized tuberculosis, lesions being particularly found in the lungs and pleura, and also many small tubercles in the pia mater. He was greatly astonished. when he examined the brain more closely to find a live and viable *Cysticercus bovis* in the *pedunculus cerebri*. He then examined the carcass more minutely for further *cysticerci*, but found no more cysts. It is not likely that the presence of the single *C. bovis* in the brain was responsible for the peculiar symptoms, nor did Hoefnagel suggest that this was the case.

Serological tests have been tried in bovines, but in general they have not been considered to be specific. Clarenburg (1932) records a successful complement fixation test on an experimental calf. As antigen he used an alcoholic extract of *T. saginata*. During the first month of artificially infecting his calf he obtained a positive reaction, whereas negative reactions were obtained with the blood sera of all non-infected calves.

The diagnosis of *C. bovis* is comparatively easily made on meat inspection. Like in the case of *C. cellulosae*, the following conditions may, occasionally, be mistaken for *C. bovis*, in the degenerated state especially.

1. *Cysticercus tenuicollis*. Armed *cysticercus*. (See differential diagnosis of *C. cellulosae*.)
2. *Echinococcus cysts*. (See differentiation diagnosis of *C. cellulosae*.)
3. *Sarcocystis blanchardi*. [See differential diagonsis *C. cellulosae*, sarcosporidia (*S. miescheriana*).]

4. Actinomycotic nodules. (See differential diagnosis *C. cellulosae.*)
5. Small tubercules. (See differential diagnosis *C. cellulosae.*)

The living *C. bovis* can hardly be mistaken for any other parasite, especially if the scolex is examined microscopically. The four suckers and the absence of a rostellum and hooklets are the most notable features. Also note the calcareous corpuscles, characteristic of tapeworm tissue.

CYSTICERCOSIS IN CALVES.

The incidence of *Cysticercus bovis* in calves is not high in South Africa, judging from observations at our abattoirs. The extent of infection may, of course, be considerably higher than is anticipated, due mainly, it is believed, to the fact that calves are seldom slaughtered after six weeks old. Then again, South Africa is not, to any extent, a veal consuming country.

From the two principal abattoirs of Natal, however, quite startling reports of the incidence of *C. bovis* among calves have been forwarded.

The Manager of the Pietermaritzburg abattoir writes:—

> "An aspect of measles infection which is puzzling, is the number of calves found to be infected. I sometimes wonder whether this may not be due to the fact that while cows are driven to their grazing ground away from human habitations, the calves are kept back and often allowed to wander about in the vicinity of the native quarters, etc. The following figures show the number found to be infected at this abattoir during the past five years."

Maritzburg Abattoir, Incidence of C. bovis *in Calves.*

Year.	Calves slaughtered.	Number infested.	Percentage.
1931–32	559	31	5·54
1932–33	552	34	6·15
1933–34	670	37	5·52
1934–35	624	28	4·48
1935–36	673	47	6·98

Mr. W. A. Dykins, M.R.C.V.S., Director, Municipal Abattoir, Durban writes:—

> "We deal with about 5,000 calves per annum, and I would say that 2 per cent. are infected with these lesions (measles), but odd consignments show almost 100 per cent. infection."

In Kimberley slaughter of calves takes place between the ages of six to twelve weeks. Although a strict watch has been kept, no trace of *C. bovis* has been found among calves.

The Superintendent of the East London abattoir reports:—

> "No case has been observed in this abattoir since it was opened; the reason for this may be the fact that calves slaughtered here are very seldom older than seven to ten days."

In Port Elizabeth and in Cape Town no cases of *C. bovis* have been found in calves under six months old. Similarly, during two and a half years' close inspection at Bloemfontein, we found no cases.

In Pretoria only two cases of *C. bovis* were observed in calves, during the past five years.

Col. J. Irvine-Smith, M.R.C.V.S., supplies the following table, which shows the very light incidence of cysticercosis in calves at the Johannesburg abattoir.

Year.	Number slaughtered.	Number infected.	Percentage.
1931–32	12,585	9	0·072
1932–33	12,999	5	0·038
1933–34	14,941	15	0·100
1934–35	15,538	5	0·032
1935–36	16,763	3	0·018

It will thus be seen that the recorded incidence of *C. bovis* is extremely low in calves in the Union of South Africa, with the exception of Natal, where it is remarkably high. No information on this subject was sought from the smaller abattoirs, where the amount of veal slaughtered would be very small, and the incidence of measles in calves would be, presumably, negligible.

The incidence of *C. bovis* in calves is very high in Kenya Colony. At the Nairobi abattoir in 1935, 94 calves were condemned for *C. bovis* out of 537 slaughtered. (17·5 per cent.)

Writing from Kenya, Daubney (1936) has shown that hand-reared calves have frequently been infected from the hands of attendants who have carried *T. saginata*. Ova of the *Taenia* can very easily obtain lodgment under the finger nails of an infected person, and thus be conveyed directly into the calf's mouth, in hand-rearing.

In Europe, measles are commonly found in calves. In several countries the statistics show comparatively high infestations.

In Holland during 1930, the following percentages measles were recorded:—

The Hague	1·58% in "grazing calves" (*graskalveren*).
Amsterdam	0·04% in "fat" calves (*vette kalveren*).

(Reference: *Tijdschr. v. Diergeneesk.*, 59, *p.* 51.)

Leiden	Living *C. bovis* in 1 "grazing" calf. Dead *C. bovis* in 10 "grazing" calves.

For the years 1933 and 1934, Professor C. F. van Oijen of Utrecht supplied the following information:—

	Infections 1933.	*Infections* 1934.
" Grazing " calves	796	984
" Fat " calves	19	16

Rheeden	In 1934 1 " fat " calf was infected.
Arnhem	6 " grazing " calves (2·3%) and 1 " fat " calf were infected.
Utrecht	67 " grazing " calves were infected.
Apeldoorn	4 calves were infected .
Amsterdam:	
1st quarter:	" Grazing " calves, 0 living; 5 cases or 0·4% dead measles.
2nd quarter:	" Grazing " calves, 1 case living measles: 0·28%.
3rd quarter:	" Grazing " calves, 2 cases living measles: 0·2%, and 3 cases dead measles: 0·3%.
4th quarter:	" Grazing " calves, 9 cases living measles: 0·59%, and 30 cases dead measles: 1·9%.
Nijmegen	(1935) (Reference *Tijdschr. v. Diergeneesk*, 63 (19). 2 out of 2,929 " grazing " calves measly (pp. 1135-36.)
Leeuwaarden	(Reference, *Idem*, pp. 1135-36.) (1935) 60 calves (1·49%) measly.

For Denmark, Elvinge (1929) gives a summary of infestation in calves, in the abattoir at Odense:—

1927: 0·32% calves showed degenerated measles, 0·12% live measles.

1928: 0·58% calves showed degenerated measles, 0·14% live measles.

1929: 0·91% calves showed degenerated measles, 0·20% live measles.

Elvinge notes that the incidence of measles in calves was increasing. The average for the three years was 0·72 per cent., whereas in 1922 it was only 0·06 per cent.

According to Dickoff (1931) the incidence of *C. bovis* was very high in Bulgaria among calves, at that time. In the District of Schumen it was 5·8 per cent. The high percentage among calves could be attributed to the fact that calves were allowed to wander about the farm-yard, and easily picked up human excretum, since few Bulgarian farms had W.C. accommodation.

According to Nakanishi (1926), the incidence of *C. bovis* in calves was 37·5 per cent. in Korea. Nakanishi found 153 calves out of 408 to be infected.

Dr. Mohler, Chief of the Bureau of Animal Industry, United States Department of Agriculture, kindly supplied statistics which showed a very low incidence of *C. bovis* in calves in the United

States for the period 1926 to 1935, inclusive. According to these statistics, the average infection is about 20 per annum, out of approximately five million calves slaughtered.

A Review of a few Case Records of Cysticercosis in Calves.

Sandig (1924), Haas (1928) and other writers recorded cases of intra-uterine infection in calves.

Haas (1928) described a case of generalized measles in a calf, three weeks old. He found quite a number of cysts in the lungs, of which quite a few were transparent, while others were hardened in a capsule, which, if incised gave forth a yellowish fluid. In some there "was even a caseous mass". The cysts were slightly smaller than a pea. Apart from the lungs, Haas also found measles in all skeletal muscles, the heart and in the external and internal masticatory muscles. The opinion of the authorities at the Veterinary School at Albert was to the effect that infection must have been intra-uterine.

Brügemann (1928) found a case of generalized measles in a calf four weeks old. Apart from the heart, measles were found in the abdominal muscles, internal masticatory muscles, external masticatory muscles, shoulder muscles, etc. Altogether Brügemann found about 200 measles in this case. The calf was fed on milk only, in the stable.

Holtz (1929) found two cases of cysticercosis in "fat" calves, closely after each other, although up till then such cases had seldom been found. In the first case, a calf about 10 weeks old, he found cysts in the heart and in the pillars of the diaphragm. In the second case, encountered 8 days after the first, he again found several *cysticerci* (both viable and degenerated). Holtz discovered that both calves came from the same farmer, from whom he instituted enquiries. The calves were kept in a stable and fed on milk. This particular farmer had been treated for *Taenia saginata* four years previously. On the farm a water-closet was used, which emptied its contents onto the lands. Holtz came to the conclusion that the milk bucket, which had been used for the feeding of the calves, must have been rinsed in the water furrow which conveyed the deposits from the W.C., and that thus segments or ova must have reached the calves.

Dräger (1929) found a nine weeks old "fat" calf heavily infested. The measles varied in size from that of a wheat seed to that of a pea. Most of them were dead, but quite a few were alive. Dräger mentioned that this case somewhat contradicted the old view that only in old measles would degeneration occur.

De Vries (1930) at Haarlem, found a heavily infested "fat" calf, four months old, and mentioned that he had found one the year before, as well. Up to that time measles in "fat" calves was considered a rare condition. In both calves he found the heart heavily infested, but all measles were of the same size and were more or less uniformly distributed throughout the musculature. The specimens were about 5 mm. in size, and were, therefore, not quite full grown.

Messner (1931) described a few cases of *C. bovis* in three weeks old calves, the nature of which led him to believe that the cause of the heavy infestation could only have been due to direct infection from a *Taenia saginata* carrier. Infection could have been carried over in milk-pails, or through the carrier's fingers causing contact with the calves' mouths.

(It is improbable that more than one calf would, coincidentally, be infected intra-uterine from different mothers. The fact that the calves were only three weeks old, and that a heavy infestation was actually visible, makes one believe that infection *might* have been intra-uterine, since beef measles are usually observed at 6 weeks in meat inspection.)

Some of the sources of infection in calves have been mentioned in the foregoing review of case histories.

It might be mentioned that the South African counterpart of the Dutch " graskalf ", or " grazing " calf is seldom slaughtered at our abattoirs. Usually sucking bull calves are slaughtered at periods from a few weeks old to about four months old. If calves are weaned and turned out to graze, their ultimate destinies are usually those of milk cows, or in the case of males, those of trek oxen, or ranch oxen, and eventually they may reach the abattoir, in a fairly advanced adult stage. Hence, we are more liable to find infection in a small percentage of cases in sucking calves, and in the great bulk of cases in full grown animals.

As a summary, the origin of infection in young calves in South Africa may be ascribed to the following factors:—

1. Direct infection from a tapeworm carrier. This,' one should imagine, is a fairly common source of infection in South Africa. Native attendants, by coaxing calves in cases of hand-rearing, may easily convey infection by ova on their fingers, direct into the calves' mounths.
2. Isolated cases, such as the case described by Holtz in Holland, in which drinking utensils might have come into contact with ova or proglottides voided by a carrier.
3. Deliberate defaecation in calf kraals, by carriers. This factor needs little elaboration upon. Native servants on farms will readily use, equally, a pig sty, cattle kraal, a stable or a calf kraal for defaecation.
4. In native habitations in South Africa, it is the usual practice to drive cows away from the *stads* or kraals during the day, to their grazing. Calves remain behind and pick up whatever " succulent " material they possibly can find around the huts. The native's sense of hygiene is not over-developed, and frequently he uses the rear of his hut, or the kraal itself, to relieve himself. Either the fowl, the pig or the calf acts as a scavenger. The Superintendent of the Pietermaritzburg abattoir considers this the most probable source of infection in Natal.

NATURAL INFECTION OF THE ADULT BOVINE WITH CYSTICERCOSIS.

Environment and physical conditions play a large part in the natural mode of infection of the adult bovine.

Whereas in Europe and in some parts of Asia (e.g., Bali) floods must be considered as the premier disseminators of *Taenia saginata* eggs, it is felt that in South Africa these factors are less responsible. In fact, some abattoir observers believe that measles is far more frequently encountered during, or just after severe droughts. During the severe droughts, such as those we experienced in South Africa in 1933 and several years previously, natural grazing was reduced to a minimum, and the probability that bovines would freely ingest human excretum was greater.

Theoretically and practically it is accepted that moisture is the most important factor in the viability of all helminth ova. On the other hand, it has not yet been established how long a pasture will remain infective with *taenia* ova; what amount of drought the *T. saginata* ova will withstand, and whether bovines can freely become infected when grazing on pastures under conditions of drought. The present writer does not hold a somewhat dogmatic view that the *T. saginata* ova can necessarily withstand excessive drought ,and that grazing on drought-stricken veld is more likely to cause infection than on green, rain-soaked pasturage. The latter condition will certainly maintain the vitality of the ova.

I am, however, of the convinced opinion that in South Africa cattle will more readily ingest human excretum during times of drought, than during periods of plenty.

It will be noticed in the map and survey of the incidence of measles in Part II of this work, that the incidence of measles is relatively much lower in those areas, e.g., the Vryburg District, where wide open ranges exist as cattle runs, under ranching conditions, than in the areas where cattle are customarily brought in to human habitations at night. Under such ranching conditions, even in times of drought, there is more available grazing and less opportunity for contact with groups of humans. It may be possible that any *Taenia saginata* ova will die off quickly on such ranges, unless, of course, the humanly deposited faeces are ingested soon after excretion. The chances that human excretum will be ingested by bovines are, therefore, considerably less on vast cattle runs.

Conditions of drought leading to the ready ingestion of human excretum are of greater importance close to human habitations. This is particularly noticeable in areas occupied by natives, for example in our Native Reserves, where all land is " common property " and unfenced and consequently badly " trodden out ". Most natives in the Reserves bring their stock to cattle-posts at night. The cattle are kraaled overnight and let out early next morning. Having been kraaled without food during the night, the hungry bovines (herds consisting of milk cows, dry cows, numerous bulls and tollies, all mixed) will snatch up whatever " luscious-looking " material may be lying about the *stad*, and this frequently contains human excretum. Often some green grass may grow in the vicinity of water-holes at the cattle-posts, and any bush or grassy cover near these water-holes is used by the herd boys and women water-carriers for defaecation. As a rule this will be the only grass available near the *stad*, which is generally trodden quite bare. The African native will defaecate anywhere within his *stad*, his cattle kraal, close to his water-holes or on the nearest fringe of bush surrounding the *stad* or the cattle-post. It can be assured that he will not go much beyond the first fringe of bush.

According to older writers, Leuckart, Neumann and others, in Abyssinia, where a very high incidence of *C. bovis* formerly existed, and where the incidence of *T. saginata* was almost 100 per cent. among the natives, very similar conditions existed. The hygienic customs of the African natives are similar from the Cape to the North Coast of Africa. Their primitive methods of cattle husbandry are also, more or less, uniform throughout the African Continent. Thus, Daubney (1936) relates an almost identical source of infection in Kenya. He writes that experience shows that in Kenya measles infestation is contracted largely in the neighbourhood of the homestead buildings, or at other places where natives are concentrated. Night *bomas* (the equivalent of our kraals) are frequently constructed near the homestead and are semi-permanent structures, complete with one or two mud huts of the Masai type, in which the herds and their families sleep. Any grass or bush in the immediate neighbourhood of the *boma* is used as cover by the natives during defaecation, " until eventually the whole area becomes heavily contaminated with viable tapeworm eggs." Each morning the cattle leave the *boma*, and after having been shut up all night without food, they eagerly snatch up a few mouthfuls of grass immediately they leave the enclosure. " It is here that most infestations are contracted; wide ranges for grazing during the day considerably reduces their chances of picking up eggs voided by one or two native herds."

Dr. Mönnig, at the International Hygiene Conference at Johannesburg in 1936, correctly referred to the fact that whilst man in civilised communities has done almost everything in his power to safeguard his own person from contraction of *T. sagniata* and *T solium*, through the agencies of meat inspection, very little has been done in the way of educating the farmer and the native in safeguarding his bovine or his pig from the converse infection. " We know little about the viability of tapeworm eggs under natural conditions, how long a pasture may remain infected, and by what agencies (flies, dungbeetles, birds, etc.), tapeworm eggs may be spread." (Mönnig, 1936.)

The Manager of the Pietermaritzburg abattoir supports my view that times of drought are the most favourable for the natural ingestion of tapeworm eggs, by the bovine. He writes (27th October, 1936):—" I believe that a drought has the effect of increasing the number of animals to be found infected with measles. This may be attributed to the fact that animals are forced, through shortage of food, into grazing in areas adjacent to native kraals, etc., where they would not graze in normal times. An increase in the percentage of cattle infected has been noticeable at this abattoir during periods of drought in the past."

(One of the local butchers, who at one time was a big loser through condemnations of beef carcasses for measles, recently, in conversation informed me that during the great drought of 1933 he actually saw cattle eating human excretum in the Thaba 'Nchu Native Reserve, where, at that time, not a blade of grass was to be seen.)

In other parts of Africa, e.g., in Senegal, Teppaz (1923) states that at Dakar he observed more cases of measles among lean cachetic cattle than among stock in good condition. Teppaz also mentions that he ascribes the high percentage of cases in cattle in Senegal to the fact that the Senegalese graze their cattle on the excretum dumps of the towns, where little grass grows, and cattle are compelled to gnaw the ground. It would appear that the Senegalese use any unfenced ground for defaecation.

From Asia Minor, Valade (1927) records that the sanitary customs of the Syrians are equally disgusting. Human excretum is dumped at random around the towns.

In South Africa there have been no records that *C. bovis* has been contracted on sewage farm pastures. In some other countries mild outbreaks of *C. bovis* have been recorded, as a result of pasturage on sewage farms. The only outbreaks of *C. bovis* infection, of any importance, in Australia occurred a few years ago among cattle which had been pastured on the Werribee sewage farm in Victoria. According to Mr. J. Drabble, B.V.Sc., Veterinary Officer in charge of meat inspection at the New South Wales State Abattoir, when the outbreak at Werribee was reported in the Press, the public of Victoria refused to buy beef. This caused a good deal of consternation among cattle owners, and the Government had to assure the public that cattle from the sewage farm would, in future, be slaughtered and utilized for purposes other than human consumption.

In Germany and in Holland there appears to be a good deal of difference of opinion as to whether pasturage on sewage contaminated lands (including the feeding of cattle with hay and other fodder grown on such lands), or whether pasturage on flood-water lands is the greater danger of infection of bovines with *C. bovis*. Among writers who held the opinion that sewage contaminated pasturage was the greater danger were Zwijnenberg (1920), K. Müller (1927), Wernery (1931), Krueger (1934 and 1935), and also Dr. Müssemeier of Berlin. Among those who favoured the opinion that flood waters disseminated *taenia* ova and thus greatly contaminated grazing were Profè and also Prof. C. F. van Oijen of Utrecht.

Dr. Müssemeier of Berlin, in a personal letter (15.12.36) expressed the opinion that the feeding of bovines on hay and other cattle-fodders grown on *Rieselfeldern* was the greatest source of infection in Germany. He defined the term "*Rieselfeldern*" as "those lands which are flooded with city drainage waters (*Abwässern*), which may even contain human faeces." (In other words sewage contaminated lands.)

Zwijnenberg (1920) was of opinion that the increase in the number of cases of cysticercosis in bovines in Holland and in Germany since the Great War, could be attributed to the greater amount of human faeces which were at times used for manuring grazing lands, owing to the shortage of fertilizer.

K. Müller (1927) pointed out the risk of depositing human excretum on grazing lands. Lands used for depositing human faeces should only be used for agricultural purposes. Another source of infection in Müller's opinion was the habit of some farmers to defaecate in stables.

Wernery (1931) believed that the spreading of measles resulted mainly from the grazing of cattle on lands used by humans for defaecation, or on lands on which faeces were deposited.

Krueger (1934) expressed the opinion that the chief source of infection of bovines was the grazing on lands contaminated with sewage (*Rieselfeldern*), or the feeding of stock with hay, grass and other food-stuffs grown on such lands. He mentioned that in Kottbus 190 tapeworm carriers were receiving medical attention, and that 2 per cent. of all cattle slaughtered in Kottbus were found infected with cysticercosis. Later (1935), after Profè had attacked his views, Krueger reiterated his previous remarks, and stressed the point that grass from *Rieselfeldern* was twice as effective in spreading *T. saginata* ova and thus infecting cattle with *C. bovis* as other green fodder in Kottbus.

Against the opinions expressed by Krueger, Profè (1934) wrote. He somewhat severely criticised Krueger's opinion, and maintained that Krueger had not cited sufficient proof that the Kottbus cattle were infected through grazing on the *Rieselfeldern*, or from fodder grown on such lands. Profè was of opinion that far more tapeworm eggs were conveyed in flood-waters from streams which covered grazing lands.

Prof. C. F. van Oijen informed the present writer (13/10/36) that he ascribed one of the main reasons for the large percentages of cases of *C. bovis* at some of the Dutch abattoirs, e.g., Rheeden, Arnhem, Amersfoort and Haarlem, to the fact that they were situated on, or close to, some of the large rivers. He wrote as follows:—

> "One can imagine that the water of the Rhine will become heavily infected with *taenia* eggs in the densely populated industrial areas of Germany. The Rhine-water floods the grazing of the parts where many of the stock slaughtered in the above-mentioned towns come from. In the event of the eggs not dying off (*niet te gronde gaan*), the chances of infection for these cattle are much higher. We have confirmed the bacteriological contamination of the Rhine-water by the mentioned industrial areas, deeply into our territory. It is, therefore, also probable that the *taenia* eggs may arrive quite viable, although we have no actual proofs to that effect."

Watkins-Pitchford (1923) was at least one South African writer who favoured the probability that flood waters could be considered the main disseminators of cysticercosis infection. His opinion is strongly supported by the fairly heavy incidence of measles at some of our South African abattoirs, which draw their slaughter-stock from coastal native areas (see Incidence Survey, figures for Kingwilliamstown, East London, Fort Beaufort and Port Elizabeth). Watkins-Pitchford, quoting from the Annual Report of the Director of the

Johannesburg Abattoir for the year 1922, stated:—" Bovine infestation varies according to the districts from which cattle are received: cattle from coastal areas show a greater percentage of infestation than cattle from inland districts. This peculiarity is doubtless to be attributed to the relative dampness of the pasturage and greater frequency of streams—factors which facilitate the survival and distribution of the segments and ova of the worms when passed in human faeces ".

Le Coultre (1928) attributes the very high incidence of *C. bovis* on the Balinese *sawahs* to flooding conditions. *Sawahs* are lands (rice, maize, ground-nuts, etc.) which are irrigated from the streams by ordinary damming and flooding. Le Coultre mentions the possibility that one or two tapeworm carriers in the mountains may, by defaecating in the streams, cause thousands of *taenia* eggs to be disseminated over the *sawahs*. After the harvest of the crops it is customary to graze stock on some *sawahs*. Under other circumstances stock (including cows) are used for cultivation while the crops are growing, and what little grazing they obtain, they do on the *sawahs*.

Lievré (1933) attributes the occasional heavy infestations of individual cattle in France to the ingestion of complete segments in human stools passed in stables, on grazing lands, etc.

Nielsen (1935) expresses the opinion that bovine infestations in Denmark are most frequently acquired in summer, but he cannot attribute any direct cause for that.

To summarize, the present writer is of opinion that the main source of infestation in South Africa is the native's insanitary customs. Conditions of drought undoubtedly play an important part in the propagation of this parasitic species, in so far as that under such conditions, especially in the badly trodden-out Native Reserves, hardly a blade of grass may survive in the veld. Native cattle then frequently remain in the vicinity of the *stad*, where they may still find morsels of food, whereas out of the drought-stricken veld nothing is to be found. These morsels of food frequently consist entirely of human dejecta.

On large open ranges the probability that the bovine will ingest human excretum is much less.

In the interior of the Union streams play little or no part in the propagation of *taenia* ova, since, in general, our interior streams consist of dry sandy spruits, which, more frequently than not run only after heavy rains. A greater danger, from this source, in the present writer's opinion is that on occasion a *taenia* carrier may defaecate into, or on the edge of pools (*kuile*) of standing, sometimes stagnant, water in these *spruits*. Such contaminated water may then be an important source of infection to the bovine, especially if cattle use the pools for drinking. It is extremely doubtful if flood waters are as important in South Africa, as they are claimed to be in Europe, in dissemination of cysticercosis. When our South African rivers come down in flood, the huge volume of water generally flows swiftly, between the very steep banks of our rivers. Very rarely is the country so flat that the banks are simply flooded over, and that adjoining grazing is very much affected. Direct contamination of confined areas of grazing, kraals, drinking places and occasional shortage of food are the main source of infection in South Africa.

PART IV.

A. The Judgment of Measly Carcasses.

In many countries in which the incidence of *Cysticercus bovis* is low, it is customary to condemn a measly carcass, irrespective of whether only a single measle, which may even be degenerated, or many measles may be found in the inspection incisions. In some other countries with a relatively high incidence of beef measles, it has been considered wilful waste to condemn lightly infested carcasses outright. Ways and means of sterilising such lightly infested carcasses have been found, so that after various modes of treatment the infested carcasses have been considered, or even rendered, fit for human consumption. These various methods of sterilisation, and the time required for the treatment of the carcass according to whatever method may be employed, have been based upon the results of tests for the viability of measles subjected to the various processes.

With regard to *Cysticercus cellulosae*, it has been customary in many countries to condemn measly pig carcasses outright, no matter how light the infestation of the carcasses may be. This somewhat severe judgment, it is supposed, has been based on some of the erroneous opinions of many of the older writers who considered, e.g., that the pig measle was not destroyed by freezing, or correctly so, that the older chilling method of sterilisation had comparatively little effect on the *C. cellulosae*.

Furthermore, it was considered uneconomical to treat measly pork carcasses for definite (formerly prolonged), periods in freezing chambers. The last named is probably the reason why few abattoirs in South Africa encourage the treatment by freezing of measly pork carcasses.

JUDGMENT OF MEASLY CARCASSES IN GREAT BRITAIN.

According to Leighton (1927) amongst the English recommendations are:—

Section V.—Instructions as to the action to be taken in the event of evidence of other disease being found in carcasses of bovines, swine, etc. (other than tuberculosis).

A. The entire carcass and all the organs shall be condemned if evidence of any of the following conditions is found:—

(Amongst others):

6. *Cysticercus bovis* (measly beef), if generalized in the meat substance.

7. *Cysticercus cellulosae* (measly pork), if generalized in the meat substance.

With regard to the judgment of measly carcasses in Scotland, Leighton (1924) quotes:—

> "In the event of evidence of *Cysticercus bovis* (beef measles) being found in a carcass and/or in a head, the carcass and/or the head may be passed for human consumption provided that they are placed in cold storage at a temperature not higher than 20° Fahrenheit, for a period of at least three weeks, or, alternatively, they shall be seized." The Section is, of course, devised to permit of saving such measly carcasses slaughtered at abattoirs where suitable refrigeration is available, since it is believed that a temperature of 20° F. for three weeks is lethal to the *Cysticercus bovis*.

Section A of Part V of the Scotland Meat Regulations (1924) provides that the entire carcass and all the viscera of pigs infected with *Cysticercus cellulosae* shall be condemned.

THE JUDGMENT OF MEASLY CARCASSES IN GERMANY.

In the fourth (English) edition of his "Handbook of Meat Inspection", von Ostertag (1913) gives the following official Regulations concerning the method of procedure with measly hogs up till that time.

For the Kingdom of Prussia, the following Ordinance was passed on February 16th, 1876:—

"1. That fat obtained from measly hogs by rendering or cooking may be utilized unconditionally, but that lean meat can only be admitted for sale or for use in one's own household in cases where it is only slightly infested with *cysticerci* and is thoroughly boiled under police supervision after having been previously cut up. (According to a decision of the Second Criminal Senate of the Imperial Court, March 25th, 1884 (p. 106), the rendered fat of measly hogs is to be sold under declaration.—von Ostertag).

2. That no objection whatever, from a sanitary police standpoint can be raised against the use of suitable parts of measly hogs in the preparation of soap or glue, or against the free utilization of the skin and bristles, and the chemical utilization of the whole body, and that these uses are to be permitted without hesitation."

3. That in all cases in which hogs are found to be badly infested with *cysticerci*, care must be exercised by the police to secure the certain destruction of the carcass, after this has been utilized as far as possible.

With reference to the utilization of viscera free from *cysticerci*, a decree of the Ministeries of the Interior and Education, June 26th, 1883, permits the fat, liver and intestines of hogs found to be measly to be freely admitted to the market as food for man, provided they have been found, upon examination, to be free from *cysticerci*. Von Ostertag (1913) gives the following Regulations, in accordance with the opinion of the Royal Superior Medical Committee, May 20th, 1882, which were applicable to Bavaria:—

1. The meat of hogs extensively infested with cysticerci is to be withheld from consumption and from the public market and is to be rendered harmless in a suitable manner. In the case of fat hogs, the separation and removal of the bacon is to be allowed at the request of the owner. No objection can be raised to the technical(¹) utilization of such animals.
2. In cases where the *cysticerci* occur only sparingly in the meat, it may, according to(²) the opinion of a scientific meat inspector, and after it has been properly cooked under police supervision, be turned over to the owner for use in his own household.

   The owner is to be properly instructed concerning the danger to human health from measly meat and is to be made cognizant of the police regulations concerning the control of such matters.
3. The public sale of meat, slightly infested, is to be permitted in *freibanks* under declaration of the danger from the meat only after it has been properly cooked under police supervision.

In the Kingdom of Saxony, the meat of hogs slightly infested with cysticerci is to be admitted to the market in a cooked or pickled condition, as unmarketable. The fat may be treated by rendering instead of boiling or pickling. The liver, spleen, kidneys, stomach and intestines of measly hogs may be utilized in a raw condition as non-marketable, provided they are found to be free from *cysticerci* by veterinary inspection.

In his work of 1934 von Ostertag gives the following directions for the treatment of pork or meat of other animals (canines) infected with *Cysticercus cellulosae*:—

1. In the dog, the whole carcass is unfit for food.
2. In a mild infestation in swine, the flesh is fit for food when cooked, steamed or pickled but not when chilled or frozen.
3. The fat of infested swine is fit for use.
4. The cooking and steaming of cysticercous pork is sufficient when the innermost parts are grayish-white and there is no red meat juice.

(¹) and(²) The wording is precisely as that of von Ostertag.

(¹) It is not clear what is meant by "technical utilization", but it is presumed that von Ostertag interprets the Regulation to mean that the meat of measly hogs may be used as fertilizer, meat meal and for other technical purposes, but not for human consumption.

(²) The wording of this Regulation is not quite clear, but, here again, it is presumed that von Ostertag infers that, if in the opinion of a qualified meat inspector (presumably a veterinarian) the carcass is not too grossly infested, the owner may receive it for his own use, but not for sale.

For measly cattle, in his " Handbook of Meat Inspection ", 1913, von Ostertag states than in the Kingdom of Prussia the method of procedure with the meat of measly cattle was regulated up till 1913 by a ministerial decree of November 18th, 1897, which read as follows :—

> " Since the conditions for the destruction of the beef measle worm have been more accurately determined by detailed investigations, than in the case of the pig measle worm, we have compiled ' the principles for the sanitary police procedure with measly cattle and calves.' While we hereby repeal all previous regulations and order that until further notice, procedure in this case shall be governed according to the principles hereby formulated. We call attention at the same time to the following statements :—
>
> Meat is to be considered well boiled, when a uniform gray colour is observed on a fresh cross section.
>
> The content of salt solution is to be accurately determined or controlled in the preparation of brine, or by means of the alkalimeter.
>
> The pieces to be utilized in pickling shall not be heavier than 2½ Kgm.
>
> Pickled meat is to be kept under police control during the prescribed period.
>
> For the determination of the temperature in cold storage rooms in operation in public abattoirs, tested maximum and minimum thermometers are to be used, and reliable self-registering hygrometers for the determination of the moisture.
>
> The temperature and moisture content of the room are to be taken during the forenoon and evening of each day and to be registered in tabular form.
>
> When properly equipped, cold storage rooms in operation in public abattoirs can be considered as ' suitable '. The district veterinarian, in co-operation with the local police authorities, shall decide in each individual case whether the conditions for the proper treatment of the meat by cooking or hanging are present. The meat of cattle which are only slightly infested with *cysticerci* may be hung in quarters in special apartments under police control; that of calves in a similar condition, without quartering. In a given apartment, only the meat of one or several measly animals slaughtered on different days should be placed in the same apartment, only when the pieces of meat are so stamped that all possible confusion is avoided.
>
> Although it has been demonstrated by previous investigations that decomposition of meat does not take place in cold storage rooms with the required temperatures and moisture content, it should, nevertheless, be determined by a veterinarian after the lapse of 21 days and before the meat is discharged, whether the meat has kept well and is not tainted.

By means of the provision that the meat of animals slightly infested with *cysticerci* and which has been rendered suitable for human consumption, shall be sold only to the consumer or for domestic use, it is intended to prevent commercial middlemen, butchers, sausage makers and hotel keepers from obtaining possession of such meat. If considered necessary, the resale of this meat is to be forbidden under the penalty of law."

Von Osterdag then proceeds to quote the Principles governing the Sanitary Police Procedure with measly cattle and calves:—

According to the number of *cysticerci* found in the routine incisions, distinction is made between—

(*a*) animals with at most ten living *cysticerci*: *slightly infested animals*;

(*b*) animals with more than ten living *cysticerci*: *heavily infested animals.*

For free utilization as human food are admitted—

(1) rendered fat, unconditionally;

(2) the liver, spleen, kidneys, stomach and intestines of animals slightly infested with *cysticerci*, in so far as these organs are found upon veterinary inspection, to be free from *cysticerci*;

(3) animals slightly infested with *cysticerci* in which the *cysticerci* which are found, are according to veterinary opinion, in a condition of complete calcification.

It is permitted to sell meat of animals slightly infested with *cysticerci*, after its dangerous properties have been removed under veterinary supervision, for domestic use, or for sale in special booths, *freibanks*, etc., in pieces not larger than 2½ Kgm., and for sale only to the consumers and under statement of its measly nature.

The necessary treatment required is—

(1) thorough boiling, or,

(2) pickling for twenty-one days in 25 per cent. brine solution, or,

(3) preservation for twenty-one days in suitable cold storage rooms in which a temperature of 3° C. to at most 7° C. prevails, and a moisture content of 70 to at most 75 per cent.

The carcasses of animals badly infested with *cysticerci* are to be utilized for technical purposes, or otherwise rendered innocuous under police supervision.

For the Kingdom of Saxony, von Ostertag (1913) states that the meat of measly cattle, according to Section 5 of the New Regulations, Appendix 6 to Section 16 of the Regulations for carrying out the Saxon Meat Inspection Law (principles underlying the judgment of meat), is to be thoroughly boiled, pickled or refrigerated.

In the Grand Duchy of Baden, the following principles prevailed, prior to 1913 (von Ostertag, 1913):—

1. Meat is to be considered as unfit for food, when the *cysticerci* are present in such number that they are seen on the majority of the cut surfaces in the body musculature.
2. The meat of animals slightly infested with *cysticerci*—that is, animals in which only isolated *cysticerci* occur, except in the muscles of mastication—is to be considered as fit for food, but not marketable, after a previous boiling, pickling or refrigeration for three weeks under police supervision. The temperature in cold storage must not exceed 5° C. If the *cysticerci* are shown to be dead, this procedure is not necessary.
3. The meat of animals in which only isolated *cysticerci* occur in the muscles of mastication is marketable, but in such cases the head is to be treated according to No. 2.

Von Ostertag (1934) gives the following summary of the revised German Regulations for the treatment of beef infested with *C. bovis*:—

1. *Severe Infection.*—Living or dead *cysticerci* found in the majority of the seats of predilection and other muscles after incisions in more than one place; or a watery or discoloured condition of the flesh, without reference to the degree of cysticercous infection.

   Judgment.—The whole carcass is unfit for human consumption, with the following exceptions—fat, liver, spleen, kidneys, stomach, intestines, brain, spinal cord and udders, provided they are free from *cysticerci* after careful inspection, otherwise they are unfit, except the fat.
2. *Mild Infection.*—All cases in which living *cysticerci* are found, excluding cases of severe infection, and cases with watery or discoloured flesh.

   Judgment.—The whole carcass is fit for human consumption when the flesh has been pickled or kept in a cooling or freezing room for 21 days, and the *cysticerci* thereby rendered innocuous. Fat, liver, spleen, kidneys, stomach, intestines, brain, spinal cord and udders are fit, provided they are found free from *cysticerci*; otherwise they are to be treated as other parts of the body.

According to von Ostertag (1934), the following official directions are in force in Germany at present, for the preservation of beef with slight *cysticercus* infection, for twenty-one days in a cooling room or in a freezing chamber:—

A. 1. The meat must be cooled to air temperature and its surface well dried in air before it is placed in the cooling room.
   2. The infected meat must be kept separate, under lock and key, from other meat.
   3. The day of introduction into the cooling room must be clearly marked on each portion of meat.

4. The separate parts or quarters of the animals must be hung so as to be exposed to air on all sides. The abdominal integument must be extended so that it does not lie upon other parts of the flesh.
5. The temperature in the cooling-room must be kept at 0° C. to plus 4° C. The humidity of the air should be:—

   At plus 4° C. not more than 75 per cent.
   At plus 3° C. not more than 78 per cent.
   At plus 2° C. not more than 81 per cent.
   At plus 1° C. not more than 85 per cent.
   At 0° C. not more than 88 per cent.
6. The humidity is to be registered by a self-regulating hygrometer, which must be tested from time to time.
7. Meat which has been kept in the cooling-room for twenty-one days, must not be sent to market till it has been certified by a veterinary inspector as of good quality and free from taint.

B. 1. Before the meat is placed in the freezing-room it must be cooled to air temperature. When a cooling-room is at hand, further cooling to about plus 5° C. is suitable.

Directions 2, 3, 4 and 7 are the same as for the cooling-room.

5. The average temperature of the freezing-room should be at least -6° C. to -8° C.
6. The frozen meat should not be cut up before thawing, but should be thawed " in the piece ". Any mould present on the surface should be removed with a knife before thawing.

The best temperature for thawing is from plus 5° C. to plus 6° C., and a humidity of 75 per cent.

Buri (1915) proposed a scheme for the judgment of measly beef, applicable to Switzerland, which coincided almost identically with that in use in Germany at that time. In this scheme Buri discriminated between " single measled ", " multi-measled " and " heavily measled " bovine carcasses.

(According to von Ostertag, quite a number of workers in Germany and elsewhere, amongst whom were Müller, Noack and Lauff, raised objections to the detention for treatment of " single-measled " carcasses, especially those in which no further *cysticerci* were found after careful search.)

JUDGMENT OF MEASLY CARCASSES IN HOLLAND.

In Holland, the Netherlands *Vleeschkeuringswet* of 1919 as amended in 1922, prescribed *inter alia*:—

Lightly infected beef carcasses can be passed as fit for human consumption:—

(*a*) After sterilization, and also after the meat has been (*b*) ten days in a freezing chamber at − 10° C., or,

(*c*) has been pickled for three weeks in 20 per cent. salt solution, in pieces of 3 Kgm., or,

(*d*) has been preserved for three weeks in a chilling-room at a maximum temperature of plus 4° C.

For France, Piettre (1922) recommended any of the following modes of treatment of lightly infested measly meat. According to that writer, it would appear that up till 1922 no Regulations existed in France for the treatment of measly meat:—

(*a*) Heating; (*b*) Pickling; (*c*) Freezing; (*d*) Cooling chambers.

Referring to Syria, Valade (1927) recommended:—

(*a*) Total condemnation in cases of generalized cysticercosis.

(*b*) Total condemnation of emaciated carcasses with only a few localized *cysticerci.*

(*c*) Passing of the carcass in those cases in which only one or two measles are found in the predilection sites named by him.

JUDGMENT OF MEASLY CARCASSES IN THE UNITED STATES.

The United States Bureau of Animal Industry Order 211, Regulation 11, Section 17, is quoted by Edelmann, Mohler and Eichhorn (1934). This Regulation allows the passing for sterilization of carcasses affected with *Cysticercus cellulosae*, but if the infestation is excessive, the carcass is condemned.

Edelmann, Mohler and Eichhorn state that measly carcasses of pork are sterilized by high temperatures and strong brine solutions, but provision for the freezing of measly pork carcasses has not yet been incorporated into the American meat regulations.

> " Meat is considered heavily infested when the measles are found alive or dead in large numbers in areas as large as the palm of the hand, on incising muscles in the favourite locations of the measles. This is the case, as a rule, when in the majority of the cut surfaces more than one measle is found in each section."

The same writers quote the Bureau of Animal Industry (U.S.A.) Order 211, Regulation 11, Section 16, which deals with the judgment of measly beef carcasses:—

> " Carcasses of cattle (including the viscera) infested with tapeworms cysts known as *Cysticercus bovis* shall be condemned if the infestation is excessive, or if the meat is watery or discoloured. Carcasses shall be considered excessively infested if incisions in various parts of the musculature expose on most of the cut surfaces two or more cysts within an area the size of the palm of the hand.
>
> A carcass in which infestation is limited to one dead and degenerated cyst may be passed for food after removal and condemnation of the cyst.

Carcasses of cattle showing a slight or moderate infestation, as determined by a careful examination of the heart, muscles of mastication, tongue, diaphragm and its pillars, and portions of the carcass rendered visible by the process of dressing, may be passed for food after removal and condemnation of the cysts, with the surrounding tissues, provided the carcasses and parts, appropriately identified by retained tags, are held in cold storage, or pickle for not less than twenty-one days, under conditions which will insure proper preservation; and provided further, that if the temperature at which such carcasses and parts are held in cold storage does not exceed 15° F., the period of retention may be reduced to six days. As an alternative to retention in cold storage or pickle, such carcasses and parts may be passed for sterilization. Fats of carcasses passed for food or for sterilization under the above provisions may be passed for food provided they are melted at a temperature of not less than 140° F. The edible viscera, except the lungs and heart, of carcasses passed for food or for sterilization under the provisions of the above paragraphs may be passed for food without refrigeration or other process of sterilization, provided they are found to be free from infestation upon final inspection. The intestines, weasands and bladders from beef carcasses affected with *Cysticercus bovis* which have been passed for food or for sterilization may be used for casings after they have been subjected to the usual methods of preparation and may be passed for such purpose upon completion of the final inspection."

JUDGMENT OF MEASLY CARCASSES IN CANADA.

The Canadian "Meat and Canned Foods Act" of 1924 as revised 29th March, 1932, provides for the treatment in cold storage for twenty-one days of carcasses slightly affected with *Cysticercus bovis.*

Section 15 prescribes that such carcasses must be reported on a prescribed form, and must be re-inspected on the day they are taken out of cold storage, and if then condemned, they must be certified on another form as "condemned on re-inspection".

In Australia no regulations for the sterilization of measly carcasses exist. According to Drabble (1936), pig measles has never been found in that country, and also, according to personal advice from that author (1936), "Wholesome meat is cheap and plentiful in Australia, and the public will not buy frozen measly meat."

The incidence of measles in Australia is so low, that the economic significance of destruction of the half-a-dozen, or so, measly bovine carcasses which have been found from time to time, has been negligible. It has been considered justifiable to condemn any carcass which might show a single measle.

JUDGMENT OF MEASLY CARCASSES IN SOME PARTS OF AFRICA.

Madagascar.

On the island of Madagascar, according to Poisson (1929), " a pig affected with cysticercosis, whether seriously or not, is not to be delivered for alimentation until it has been cut up in pieces weighing about one kilogramme, and boiled for three hours; this is to avoid any danger to public health."

Northern Rhodesia.

The Medical Officer of Health, Ndola writes (11.11.36):—

> " During 1933 efforts were made to reduce losses through cysticercosis by freezing, but it was found to be uneconomical, and since then all carcasses showing cysts are condemned, irrespective of the degree of infestation."

Tanganyika Territory.

The following information on the degree of infestation which justifies the condemnation of measly carcasses, has been kindly supplied by Capt. H. J. Lowe, M.R.C.V.S., of the Department of Veterinary Science and Animal Husbandry, Mpwapwa (24.10.36):—

> " In more civilized countries the presence of a single cyst would be sufficient to condemn the whole carcass, but in a country such as this, where the incidence of *C. bovis* is high, such a procedure could not possibly be enforced. As a general rule our method is to condemn only those carcasses in which more than half-a-dozen cysts can be demonstrated in two or more sites, and in other cases the carcass is sterilized by boiling and sold as cheap meat to the natives. This is admittedly not very satisfactory in that many infected carcasses are passed for human consumption after the few demonstrable cysts have been removed, but it is thought that any improvement must await the time, when by education, the natives can be persuaded to adopt more sanitary habits in regard to the disposal of excreta and general cleanliness."

Kenya Colony.

In Kenya the standard adopted in the past has been based on that formerly applied in South Africa and in certain parts of Germany. Carcasses in which less than six viable measles can be found by the meat inspector, are passed for consumption, after removal of the infected portions of meat. Carcasses in which six or more viable measles are found are condemned, and are treated in the by-products plant at the abattoir. It is proposed to tighten up the Regulations in respect of measled carcasses in the Nairobi Municipal Abattoir in the near future, and if the proposals that have been put forward are eventually adopted, a single viable *cysticercus* will be sufficient to cause the condemnation of the whole carcass (Daubney, 1936). Mr. Daubney informs me that the Medical Officer of Health (Nairobi) recently informed the Stock Owners' Conference that were the standard of inspection raised so that any animal with a single viable *cysticercus* was condemned, the percentage of condemned cattle would be increased by 4·7 in the case of grade cattle and by 7·4 in the case of native cattle.

Union of South Africa.

In the Union of South Africa, Section 115 of the Public Health Act, No. 36 of 1919, as amended by Government Notice No. 1456 of 1933, provides for the treatment of lightly infested measly carcasses in cold storage.

Paragraph 16 (2) of that Section now reads:—

> " Every carcass found to be infected with bladderworm disease (" measles " shall, together with the viscera, be condemned as unfit for human consumption and destroyed or treated and disposed of so as not to endanger health save where
>
> (*a*) during examination as aforesaid less than ten bladderworms are disclosed; and
>
> (*b*) less than six cysts are found in the carcass apart from the head, tongue, pluck, stomach and intestines; and
>
> (*c*) cold storage to the satisfaction and under the control or supervision of the local authority, and in which a temperature of or below *minus* ten degrees Centigrade is continuously maintained, is available; and
>
> (*d*) the owner or his agent in charge of the carcass requests that it be placed in such cold storage, and furnishes a written undertaking to the satisfaction of the local authority to defray the cost of so doing."

Paragraph 16 (3):—

> " If the conditions specified in paragraph 16 (2) hereof are complied with, but not otherwise, the carcass, after removal of all obviously diseased portions, may be placed and kept in such cold storage for at least fourteen days, and may thereafter be examined and passed as fit for human consumption."

Since no Regulations are framed to the contrary, and no exceptions are made in the existing Regulations, of swine carcasses, these can also be treated in the freezing chamber. It has already been mentioned that on account of economy it is not customary in most South African abattoirs to freeze measly pork carcasses, and many abattoir superintendents and laymen are under the erroneous impression that the Regulations do not provide for the freezing of measly pig carcasses.

The writer was informed that at some centres, where no freezing facilities exist at the abattoir, the local authorities, by arrangement, permit the freezing of measly carcasses in the chambers on premises of commercial firms. I know of at least one case, in which a measly ox carcass was sent from one of the smaller rural centres to a Bloemfontein commercial house for the required freezing. The carcass was then, certainly, not under the supervision of the local authority concerned, nor could that particular local authority be *satisfied* that proper freezing at - 10° C. was continuously being carried out for 14 days. Such dealings are quite illegal, because Regulations expressly read that *cold storage to the satisfaction and under the control or supervision of the local authority,* etc., must be available. Cold stores on the premises of privately-owned commercial houses cannot be controlled or satisfactorily supervised by a local authority, and particularly so if that cold store is actually situated in another town, some two hundred miles away! It might be wise if the responsible Government inspectors could investigate such malpractices and satisfy themselves that abattoirs which do not possess freezing chambers do not permit the treatment of their measly carcasses in chambers quite out of their control.

Various amendments to this paragraph caused the time specified for freezing of measly carcasses to be reduced from 84 days to eventually 14 days. This reduction of the specified time resulted from reports on various viability tests with measly carcasses which had been performed in Europe.

B. Destruction of Cysticerci in Meat.

VARIOUS METHODS OF TESTING VIABILITY OF CYSTICERCI.

Cysticerus cellulosae and *Cysticercus bovis* in meat can be destroyed, and the meat rendered suitable for human consumption by any of the following agencies, without seriously damaging the food value of the meat:—

1. By heat up to certain temperatures.
2. By pickling in certain strengths of salt solution.
3. By cold storage at certain temperatures for specific, continuous periods of time.

Other agencies, e.g. electric rays may also be mentioned, but some writers (Clarenburg, 1932, and others) have had little or no success with them.

Before discussing the various methods of destroying *cysticerci,* it will be necessary to consider the various tests which have been employed for the viability of *cysticerci.* Such tests have been used with a view to proving whether or not *cysticerci* in meat, which has been subjected to any of the above methods of rendering it fit for human consumption, have actually been destroyed in the process, or, as von Ostertag at various times, Glietenberg (1931) and others have suggested, have been rendered innocuous, although not necessarily killed.

The reaction of *cysticerci* to external conditions, or to the influence of chemical, physical and physiological agencies, have been taken as criteria of the viability of the *cysticerci.*

Mönnig (1928) very conveniently classified the agencies which caused phenomena which were accepted by various workers up to that time as criteria of the capability of development of *cysticerci.* Mönnig's classification can, therefore, ~~be followed~~ to a great extent, with the additional details of some experiments by previous and subsequent workers.

1. *Reaction to Warming.*

" This," Mönnig mentions, " was the first criterion employed, and, in combination with other methods (warming in media), is still the most important, according to many authors. Perroncito (1877), Ostertag (1897), Ransom (1914), Porter (1923) and others employed this method."

Ransom (1914), according to Mönnig, states that if the heads show no movement in the retracted state, they should carefully be evaginated by pressure, after which they will sometimes still show movement.

Perroncito (1876) isolated *Cysticerci cellulosae* from pork and placed them on a Schulze's warm stage. At low temperatures (16° C. to 20° C.) the bladderworm remained inert, but when the temperatures passed 30° C. to 35° C., fairly lively movements of the scolices and particularly of the suckers were observed. The movements became even more intense as a temperature of 42° C. to 46° C. was reached, and gradually ceased after that temperature, until at about 48° C., they stopped altogether. In 1877 Perroncito observed that a temperature of 45° C. was sufficient to kill *C. bovis.* He based his criterion on his observation that at that temperature the *cysticerci* had a cloudy appearance, no motility was noticed when they were examined microscopically, and infection experiments on humans gave negative results.

Von Ostertag (1913), in describing his experiments of 1897, states that he found warming *cysticerci* on a stage the most convenient method. " Living *cysticerci*, when heated to a temperature of 30° C. to 40° C., exhibit under the microscope active movements of the rostellum, sucking discs and other parts of the head and neck, while dead *cysticerci* remain motionless. This thermo-microscopic investigation may be undertaken conveniently in the Nuttall microscope thermostat, or in the simpler and cheaper warming apparatus for microscopic investigation devised by Kabitz and Rissling.

Porter (1923) did not accept motility of *cysticerci* when warmed to certain temperatures, as any guide to viability. She found that while some living *cysticerci* certainly did show motile powers on gradual warming of the stage, some *cysticerci* which were definitely dead, showed the same movements. Porter mentioned that some isolated cysts which she had kept in boiling water for an hour, showed motility after cooling and subsequent re-heating on a warm stage. She also found the same type of movement on warm stages with certain materials (whether these had been frozen or not), such as indiarubber, parchment, pig's bladder, silk, catgut and chamois leather. To sum up, she did not consider that any test of viability based on movement of isolated *cysticerci* on exposure to heat could be regarded as a reliable criterion of the viability or otherwise of *cysticerci* in a joint or carcass that had been exposed to freezing.

2. *Appearance and Physical Condition.*

Mönnig (1928) quotes Ostertag, Killisch, Brohmann, Glagé, Reissmann and others, who observed changes in the appearance and physical condition of *cysticerci*, at death.

Von Ostertag (1913) quotes Hartwig, who found in *cysticerci* which had been exposed to a temperature of 65° C., and was thus killed, that the scolex, which in a living condition was unusually resistant to pressure, was so soft that it could be compressed between two glass slides, like beef tallow. " This alteration must be considered as an excellent criterion of the accomplished destruction of *cysticerci* by boiling. By means of the above demonstration, Hertwig simultaneously disproved the widespread erroneous view that *cysticerci* which had been killed by boiling or roasting could be detected in eating the meat, by a crackling sound between the teeth."

Killisch (1923) and Brohmann (1924) stated that the vesicles of live *cysticerci* are glistening and pale white, offering a certain amount of resistance on pressure, while in dead specimens they are turbid and easily burst.

" In live *cysticerci*, the scolex can be fairly easily extruded on pressure between the fingers, and appears to ' swing out ' of the bladder; in dead *cysticerci*, the scolex is sticky, drawing threads and is not easily extruded, but frequently breaks. while a whitish turbid fluid exudes from it." (Mönnig, 1928.)

Annie Porter (1923) made direct observation on the physical condition of fresh *cysticerci* both macroscopically and microscopically, and carefully compared the results with those observed in *cysticerci* from carcases slaughtered at different times, and which had undergone various periods of freezing. She noted:—

(i) In the normal fresh *cysticerci* of *T. solium* and *T. saginata*, that they glistened in appearance, were whitish to pinkish in hue, firm to the touch, not easily ruptured. The fluid within the fresh, normal bladder was practically colourless, clear and contained very few cellular elements.

(ii) After three weeks' freezing of a large hind-quarter of beef, its superficial cysts might be slightly less firm than fresh cysts, but deep-seated cysts, on thawing, were practically as tense as fresh cysts.

(iii) After four weeks' freezing, and then gradual thawing, the superficial cysts showed slight change in the colour of the fluid in the bladders, though the change was rarely more than a very pale straw colour. Deep-seated cysts, or cysts well protected by fat rarely showed such change.

(iv) After six weeks' freezing, followed by gradual thawing, some of the more superficial cysts might show a pinkish tint, unlike that of the fresh bladders, as if some haemolysis had occurred within them. This was really some indication of change of physical condition; the wall of the *cysticercus* had become more porous. In Porter's opinion this was not necessarily indicative of the death of the *cysticercus*.

(v) After eight weeks' freezing, the *cysticerci*, when thawed, showed more marked colour changes. The superficial ones were brownish red, the deeper ones near bone were pinkish, and the deepest *cysticerci* or those well protected by layers of fat, still showed little change.

Porter found that cloudiness of the contents of the bladder was not necessarily a feature in dead specimens.

She also found that freezing up to three months seemed to make little difference to the morphology of the *cysticerci*. In the majority of cases the suckers of the worm retained their distinctness, the hooks of *Cysticercus cellulosae* or of *Echinococci* remained *in situ* and showed no tendency to separate, and the calcareous bodies showed no obvious signs of degenerative effects. Porter did not notice the dissociation of the calcareous corpuscles in frozen *cysticerci*, as was observed by Reissmann (1897).

Killisch (1923) also placed no value upon the casting of the hooks as a criterion of viability of *cysticerci*. He found that frequently the hooks of *Cysticerci cellulosae*, which might still be alive, although possibly damaged by cold, might be cast or loosened.

Schmey and Bugge (1931) used the demonstration of the excretory " flame " cells as a criterion of viability. Active " flame " cells were demonstrated by them up till 39 days after slaughtering.

3. *Warming in Saline and Bile Mixtures.*

A method of testing the viability of *cysticerci* by immersion in warm fluid media was first used by Perroncito (1877), von Ostertag (1897) and Glagé (1896).

These workers placed fresh, living *cysticerci* in water, which was warmed up to 37° C., or a maximum of 40° C. Von Ostertag noticed that a living *cysticercus* in this simple medium evaginated the scolex, which frequently showed lively movements.

An improvement on the earlier methods of Perroncito, Glagè and von Ostertag was effected by Franke (1914), who added various quantities of bile to the water, so as to cause the conditions to be more like those normally in the human intestine, in which the *cysticercus* had to evaginate and develop. Franke also found that active evagination of the scolices of *cysticerci* occurred in physiological saline solution, to which a few drops of ox or pig bile had been added, and which had been heated to a temperature of plus-minus 38° C.

A still bigger improvement on the Franke method was effected by Wagner (1922). He found that the most effective evagination of scolices occurred in concentrated bile solutions—50 per cent. concentrations, or stronger, at temperatures 41° C. to 42° C. Wagner also recommended the use of only ox bile for *Cysticercus bovis* tests and pig bile for *Cysticercus cellulosae*. These could be warmed on Nuttall's microscope thermostat at 37° C., gradually increasing the temperatures to 41° C.

Müller (1923) performed his tests with *C. tenuicollis* and found the most successful results by using 2 to 4 per cent. bile solution at 38° C. Killisch (1923) found ready evagination of scolices of *C. cellulosae* in 0·75 per cent. solutions of pig bile in saline. Movements could be readily seen of the evaginated scolices, when warmed from 30° C. to 49° C. and examined on Nuttall's microscope thermostat. Rhythmic movements of the head to the right and the left were visible to the naked eye, and expansion and contraction of the suckers were plainly visible.

Glietenberg (1930) used pure pig bile, undiluted, and claimed very good results.

Sachs (1931) described the following method of testing the viability of *Cysticercus bovis* by evagination tests:—

(*a*) The measle is carefully removed from its connective tissue capsule.

(*b*) The liberated measle is then placed in a shallow watch glass in fresh ox bile. (No pig bile nor physiological saline solution is used.)

(*c*) If an incubator is not available, the watch glass is floated on the surface of water heated to 40° C.-42° C., and the water bath is covered with a lid, if better results are to be expected.

(*d*) After 1 to 3 minutes the scolex is evaginated and under the microscope lively movements may be observed.

Clarenburg (1932) obtained the best results in 5 per cent. bile solution, and he found the optimal temperature to be 40° C. He gave the following table in respect of measles taken from veal which had been preserved for 38 days in a cooler (probably his results may have been even more conclusive if he had used fresh measles for this particular test for the best strength of bile solution):—

Out of 10 *cysticerci* in 100 per cent. bile, after 1 hour, 1 completely evaginated.

Out of 10 *cysticerci* in 50 per cent. bile, after 1 hour, 1 completely and 2 partly evaginated.

Out of 10 *cysticerci* in 25 per cent. bile, after 1 hour, 1 completely and 5 partly evaginated.

Out of 10 *cysticerci* in 5 per cent. bile, after 1 hour, 4 completely and 3 partly evaginated.

Out of 10 *cysticerci* in 1 per cent. bile, after 1 hour, 1 completely and 0 partly evaginated.

Clarenburg, like Diemont (1923) found that the most rapid evagination of scolices occurred when the bile solution and the glass receptacle were first heated to 40° C. before the *cysticerci* were placed in them, and the test commenced. He also found that young *Cysticerci* evaginated more rapidly (after about 10 minutes) than older *cysticerci*, which sometimes took about eight hours to evaginate.

Malkani (1933) used fresh *Cysticerci bovis* for his tests. After having removed their outer connective tissue capsules, each cyst was placed in a petri-dish containing bile diluted with distilled water. Some petri-dishes were kept at room temperatures, while others were kept at an incubator temperature of 37° C. No change was visible in the cysts kept at ordinary room temperatures. In the case of those kept at incubator temperatures, peristaltic movements were seen, during which the alternate " protrusion and retraction of the extremity bearing the scolex was very striking." Evagination of the scolex occurred within 20 hours.

Instead of using bile, various bile salts have been employed by some authors. Amongst the bile salts which have been used have been sodium glycocholate, sodium taurocholate, sodium palmitate and sodium stearate. Clarenburg obtained very little success in evaginating scolices in sodium palmitate and sodium stearate. He found that sodium taurocholate gave better results than the glycocholate, and the optimal temperature was 40° C. A 1 per cent. solution of sodium taurocholate had almost the same successful results as a 1 per cent. bile solution. He did not obtain better results by using stronger solutions (3·5 per cent. and 10 per cent. solutions). Clarenburg, therefore, maintained that a 5 per cent. bile-saline solution was the best medium for artificial evaginations of scolices.

Using sodium taurocholate solution, Malkani, on the other hand, obtained his best evaginations of scolices of *C .bovis*. By using a 1 per cent. aqueous solution of sodium taurocholate at room temperatures, no movements or evaginations occurred, but at 37° C. incubator temperature, somewhat sluggish movements resulted and evagination of the scolices in 18 hours. By using a 5 per cent. aqueous solution of sodium taurocholate, very active movements resulted in a very short time in the incubator. Evagination of scolices occurred in 29 minutes to 2 hours. By using 1 per cent. and 5 per cent. aqueous solutions of sodium glycocholate, Malkani obtained rather less successful results. In these solutions cysts usually contracted somewhat and assumed a globular appearance, usually showed no movement, and evagination occurred usually more than 20 hours after.

Clarenburg also did evagination tests in various digestive juices. He found that evaginations did not take place readily in choline and acetocholine solutions, nor in pepsin in various concentrations in 0·2 per cent. hydrochloric acid. He, however, found good results in the used pancreatic extract, trypsinogen and pancreatin.

4. *Staining Reactions.*

These were sometimes used by earlier authors (Reissmann, who showed that dead *cysticerci* took aniline stains, whereas living *cysticerci* did not), but later workers (Killisch, Brohmann) did not consider them as sure criteria, except Porter, who based almost her entire criteria on the reactions she obtained to various stains. Mönnig, in reviewing Porter's work, states: " It must be noted here that the ' dead " *cysticerci*, used for comparison in Porter's tests were boiled and, since Porter's conclusions are based chiefly on staining reactions, all other tests being regarded as unsatisfactory or indefinite, the conclusions arrived at on this basis must be read in this light, since dead tissues cannot be expected to stain like boiled tissues without further proof, and if they do not, they can likewise not be considered to be still alive."

Porter found that Delafield's haematoxylin and an acidulated solution of aqueous methyl green, particularly the latter, proved most effective. She found, for example, with methyl green solution, that dead cysts, namely those boiled for three hours, stained a deep green in the heads; cysts frozen for 22 days showed very faint green heads; cysts from a freshly killed animal remained unstained. She found that some cysts from carcasses frozen for 77 days stained very feebly, denoting, as she concluded, slight signs of life. Similarly faint stains were noticed in *C. cellulosae* from a pig frozen for 41 days.

5. *Infectivity Tests.*

These gave the only conclusive proof of vitality of *cysticerci*. Several writers, and in particular von Ostertag, have attempted to show that although *cysticerci* might be weakened owing to external influences, their power of infection to human beings has been considerably reduced.

Actual infection tests on human subjects were performed by Perroncito (1877), Zschokke (1896), von Ostertag (1897), Ransom (1914), Porter (1923), Schmey and Bugge (1930).

That infection of the human subject with *Taenia saginata* and even more so with *Taenia solium* was not without serious risk to the subject, was appreciated by several writers. Schmey and Bugge (1931) were criticised by various persons, who averred that their claim that 21 days', or even 40 days' chilling of beef was not sufficient to kill the contained measles or render them innocuous, was based on criteria obtained by artificial means, and not by actual infection tests on their own persons. They, therefore, performed infection tests on six persons, of whom three developed six *Taeniae saginata*. In the article in *Tierärzt. Rundsch.* 1931, p. 719, in which they describe their experiments, Schmey and Bugge stress the danger of actual infection tests on humans. They had, therefore, intended doing such infection tests with *C. tenuicollis* and *C. pisiformis* on dogs. In order to silence all criticism of their work, they undertook the infection tests, and remarked: "It is easy enough to utter criticism, but we wish our critics would undergo a tapeworm infection along with us, then they will change their tune."

I would not suggest that fear of infection prompted most workers between the end of last century, when von Ostertag and Zschokke performed their infection tests and quite recently, to discard actual infection tests. Franke's bile-saline method, with the subsequent improvements effected by Müller, Wagner, etc., was considered a good criterion, since conditions approached those of natural infection. Nevertheless, a certain amount of doubt must have existed as to the correctness and certainty of results obtained by those methods.

We are therefore, indebted to Iwanizky (1932), who devised a method which very nearly reached the identical to natural infection, without endangering the health of the subject on whom the test was to be applied. Iwanizky pointed out the fact that the methods employed up till 1932, to test the viability of health-damaging *cysticerci* did not come up to requirements. He, rightly, maintained that the only sure method of testing the viability of measles was by means of " infection tests " on the human subject. According to Iwanizky, even the apparently most effective method hitherto employed, namely that of Franke, had its defects, e.g., the use of pig bile instead of human bile; the use of artificially produced temperatures, instead of natural human body heat; the artificial isolation of the measles out of their capsules, and the absence of influences of the human digestive juices.

Iwanizky, according to Keller (1935) wrote:—" Even if it were possible to put aside all the defects in Franke's method, by using human bile instead of swine bile, and that first the measles could be subjected to the influence of the human stomach juices, which in practice is not as easy as it seems, the controlled results of the viability of the measles (by using such a modified method of Franke) would depend upon quite a number of circumstances, for example, on the power of assimilation of the gastric juices, on the intensity of the influence of these on the measles, on the temperature, etc.".

Iwanizky also pointed out the undesirability of self-infection tests, such as were performed on themselves and their assistants by von Ostertag, Schmey and Bugge, etc.

Actual human infection precludes the quick results sometimes necessary, since it takes a considerable time before the subject may be satisfied that he has, or has not, contracted tapeworm infection, and before he may observe segments in his stools. It has also been suggested that a measure of immunity in a subject to tapeworm infection may exist, which would negative infection tests; then again, it may be necessary for a subject to be repeatedly infected, which would lead to confusion in the results obtained, apart from the unpleasant discomfort which the subject would experience.

These were among the factors which Iwanizky considered, when he devised a new and relatively safe method of testing the viability of *cysticerci*. He removed the measles out of the muscles and sewed them into small silk bags. According to Keller, it is clear that Iwanizky did not remove the measles from their connective tissue capsules. The silk sacs were smeared with butter, placed on the back of the tongue and swallowed. Some 20 to 24 hours later, the subject recovered the silk bags in his stools, and the contents of the bags were then examined to see whether digestion and absorption, in the case of dead measles, or whether evaginated and developing scolices, in the case of viable measles had resulted. According to Iwanizky, the caudal vesicles were digested by the digestive juices of the subject, after evagination of the scolices therefrom, and the liberated scolices could be accepted as a definite criterion of viability of the future tapeworm.

Keller devised a still further improvement on Iwanizky's method, which he described in 1935. He realized that scolices evaginated or liberated from their caudal vesicles in the small intestine could be squashed inside the silk bag when passing through the large intestine. Keller explained that in the large intestine a coagulation of the contents of the intestine takes place, and the walls of the silk bags could thus be squeezed together, with resultant damage to their contents. Keller's modification consisted of placing the measles to be tested into small celluloid tubes. 10 mm. long, with an outside diameter of 7 mm. and a wall thickness of 0·5 mm. "These tubes are sewn into a stretched silk bag, so that the two open ends are covered by an even layer of silk." The interior of the celluloid tubes can be penetrated, through the taut ends of the silk covers, by the digestive juices from two sides, and the measles are "protected from outward pressure, as it were, in a small cage". Another advantage mentioned by Keller is the fact that the smooth tube and the tautly drawn (drumlike) silk ends of the tubes show no pleats, as often happens in using silk bags. He found, also, that the best results were obtained after he had carefully removed the *cysticerci* from their connective tissue capsules. According to Keller, liberation of the scolices by this method, which precludes any outside interference with the measles, must be accepted as the most satisfactory criterion of viability of measles, and he considered it quite unnecessary to do control or contemporary experiments in gall. As an absolute test of the effectiveness of his method, Keller used for one experiment, only absolutely fresh measles (from a newly-slaughtered animal). He pointed out that by his method, out of 13 measles swallowed, 10 evaginated their scolices undigested, whereas by using Iwanizky's method, out of 12 measles swallowed, 6 were digested. (Probably as the result of destruction in the intestine.)

Having considered the various methods which have been employed to test the viability of *cysticerci*, and the phenomena which have been taken as criteria that such *cysticerci* were actually dead, or else rendered innocuous, or thirdly were still capable of development, we may now continue with the discussion of the agencies which have been found to be destructive to *cysticerci*.,

1. *The Effect of Heat on Cysticerci.*

Most of the early workers realized that heat, at certain temperatures, will with certainty cause the death of *cysticerci* in meat.

The most thorough investigations regarding the power of resistance to heat, of the bladderwroms of the pig, are due to Perroncito, 1872 (Leuckart).

Perroncito was at first inclined to the opinion that it required a temperature of at least 125° C. to render the bladderworms harmless, but he was afterwards enabled, by means of a more conclusive test, to establish that the measles are certainly killed when the temperature of the surrounding fluid reaches 50° C., or even below that, and when the *cysticerci* remain in it longer than a minute. One of Perroncito's assistants swallowed several *C. cellulosae* which had been heated to 50° C., and remained free from tapeworm infection.

Pellizari and also Lewis and Cobbold opposed Perroncito's views and fixed the lethal temperature of *C. cellulosae* at 60° C. The effect of thoroughly cooking measly pork was observed by Pellizari, who showed that in Florence the inhabitants were immune to *Taenia solium*, because pork was never eaten half raw like beef, by them. and from eating the latter they frequently developed *Taenia saginata*. Marchi, according to Leuckart, 1886, and von Ostertag, 1913, only found a single *T. solium* in a certain time in Florence, out of thirty-five *taeniae* examined by him, although during that time no fewer than 13,000 measly swine had been consumed in Florence.

Neumann (1892) pointed out the practical difficulty in knowing under what conditions the centre of a piece of flesh (pork) would reach the temperature destructive to the measle. In cooking large pieces of meat, Küchenmeister had noticed that after half-an-hour, when the external temperature was 60° C., the temperature of the interior had reached 55° C.; in about three-quarters of an hour, the exterior temperature was 77-80° C., and that of the interior 63° C. Pellizari, testing measly pork, put two pieces weighing 600 grammes and 10 cm. thick, in boiling water—one piece for five minutes, the other for half-an-hour. When removed, the temperature of the former was 45·5° C. in the centre, and that of the latter 81° C. Taking into account the loss of heat by radiation, these two temperatures may be estimated at 51° C. and 83° C. (Neumann). For roasted pork, Vallin (Neumann) has found that while its external temperature necessarily exceeds 100° C., beneath this superficial zone it is " touched " by cooking; a zone beneath this again oscillates between 52° C. and 53° C., but in the centre it does not exceed 46° C. to 48° C.

With reference to *C. bovis*, Perroncito (1877) observed that a temperature of 45° C. was sufficient to kill the measles. Perroncito found that *C. bovis* was sometimes destroyed at 44° C., often at 45° C. to 46° C., and between 47° C. and 48° C. it was always destroyed. Three of his assistants voluntarily swallowed a *C. bovis* each—one measle had been heated to 47° C., and gave no signs of life; another had shown no motility at 45° C.; the third was heated to 44° C. and had shown slight motile powers. In none of the three tests did a tapeworm develop.

Claienburg (1932) found that *C. bovis* were killed within 15 minutes after immersion in boiling water.

2. *The Effect of Pickling on Cysticerci.*

Perroncito was among the first investigators who tested the possibility of destroying *cysticerci* in meat by pickling in brine. He used brine composed of 2½ parts saltpetre, 20 parts of cane sugar, 250 parts common salt, 1,000 parts water. He found that *cysticerci* contained in measly beef and pork were killed in fourteen days, provided the meat was no thicker than 6 cm., or when the brine was injected into the meat by means of a syringe. Von Ostertag (1913), in referring to his early investigations, described a process of demonstrating the completion of successful pickling of meat. He employed a 1 per cent. solution of silver nitrate, which produced no striking change on the cut surfaces of fresh muscle meat, but, on the cut surfaces of completely pickled meat, a temporary milky cloudiness was produced (chloride of silver). Glagé found that a 2 per cent. aqueous solution of silver nitrate was even more effective in this test.

Schmey and Bugge (1931) found that by using a brine-pump the time required for pickling measly meat could safely be reduced from 21 days to 7 days. They mentioned that fat was slower in the pickling process than fleshy meat.

Clarenburg (1932) noticed that *C. bovis* was killed in 5 days in solutions of 20 to 25 per cent. brine.

3. *The Effect of Prolonged Preservation in Cold Storage on Cysticerci.*

Perroncito (1877) believed that *cysticerci* would only survive for a limited time after the death of the host. In an experimental calf he found that measles were dead 14 days after the slaughter of the animal. Von Ostertag (1897) found that this was not so in all cases, and that death of the *cysticerci* did not necessarily follow within such a short period, but by preserving beef in a cooler for three weeks, the *cysticerci* contained therein would be rendered innocuous. Von Ostertag performed various infection tests on human beings, with *Cysticerci bovis*, which had been preserved for periods varying between 16 days and 21 days. His tests resulted as follows:—

1 person ate 2 measles 16 days after slaughter of animal and got 0 taeniae.

1 person ate 1 measle 19 days after slaughter of animal and got 0 taeniae.

9 persons ate 52 measles 20 days after slaughter of animal and got 0 taeniae.

31 persons ate 166 measles 21 days after slaughter of animal and got 0 taeniae.

4 persons ate 15 measles 14-19 days after slaughter of animal and got 10 taeniae.

After 21 days' preservation of the meat, in another test, he again failed to infect a man with tapeworm.

Glagé (1896) found that 15 days' preservation of beef was not sufficient to kill the measles. He swallowed three measles from such beef, and developed two tapeworms.

Zschokke (1896) infected himself with one tapeworm after having swallowed five *C. bovis* which had been preserved for 16 days in meat, after slaughter of the host. He repeated the test with five measles from a bovine carcass which had been preserved for 21 days, and failed to infect himself.

Kabitz, according to Clarenburg (1932), developed three tapeworms from three measles out of beef preserved for 15 days.

Von Ostertag (1897), and indeed until about 1930, maintained that the preservation of measly beef for 21 days was quite safe from a public health point of view, since no infection resulted from such measles, although certain movements could still be noticed under observation in Nuttall's microscope thermostat. When Müller and Wagner in 1922-23, and van Santen in 1928 disproved von Ostertag's claim that measles could not survive the death of the host by 21 days, he then steadfastly maintained that although those workers, by means of Franke's test, had caused evagination of scolices of such measles, he was not satisfied that those measles were still capable of developing into tapeworms, although the tests of his opponents might have shown that they were not dead. Von Ostertag thus discriminated between "measles killed, or dead" and "measles not actually killed, but weakened and thus rendered incapable of developing". This was considered and proved to be a very risky view, by Schmey and Bugge and by other subsequent workers.

Mönnig (1928) gives the following table showing the proportion of measles which still showed movements in von Ostertag's tests of 1897, with the number of days after slaughter of the host:—

Days after slaughter	14	15	17	18	19	20	21	22	23	24	25
Proportion showing movement........	23/41	8/12	3/10	6/12	12/29	12/68	8/71	2/10	2/9	2/16	0/16

After 18 days' cooling, van Ostertag noticed only very slight movements, and on the 19th day the vesicle fluid was opaque; on the 20th day the heads became opaque. Von Ostertag then advanced further confirmatory evidence by performing digestion tests with hydrochloric acid—pepsin and incubation at 37° C. He found that eleven cysts which had been preserved in meat for 20 days, and had shown slight neck movements on warming, were completely digested within an hour.

As the results of these tests von Ostertag maintained that the rendering of measly beef harmless by preservation in cold storage (at temperatures just above freezing), was the most rational method, since the meat thereby underwent the least depreciation in value. suffered only a minimum loss of weight and found a ready sale as raw meat. The same treatment was not applicable to *C. cellulosae,* since von Ostertag found them alive 42 days after slaughter of pig carcasses.

Franke (1914) found that after 16 days' cooling half the measles tested, evaginated the scolices, but none after 20 days' cooling.

Wagner (1922) was probably the first worker to doubt the reliability of von Ostertag's views. He found that after 24 to 26 days' preservation of beef, the measles still showed movement, and evagination of scolices still occurred in concentrated bile solutions (50 per cent. bile solutions, or even stronger).

The danger of reliance on the opinion that 3 weeks' cooling of meat would be destructive to *C. bovis,* or otherwise render them innocuous, was further pointed out by van Santen (1928). This worker found that *C. bovis* was definitely not destroyed by three weeks' preservation of the meat, and, indeed, he found that after 37 days' cooling three out of 16 measles were capable of evaginating their scolices. Van Santen employed Franke's tests (1914), viz., warming in bile solution to 37° C. He supplied the following table as indicative of his results:—

After	19	days'	cooling,	out of	16	tested measles,	9	living.
,,	20	,,	,,	,,	24	,,	17	,,
,,	21	,,	,,	,,	36	,,	24	,,
,,	22	,,	,,	,,	69	,,	48	,,
,,	23	,,	,,	,,	39	,,	19	,,
,,	24	,,	,,	,,	28	,,	25	,,
,,	25	,,	,,	,,	21	,,	10	,,
,,	26	,,	,,	,,	65	,,	24	,,
,,	27	,,	,,	,,	22	,,	11	,,
,,	28	,,	,,	,,	36	,,	3	,,
,,	29	,,	,,	,,	40	,,	1	,,
,,	30	,,	,,	,,	32	,,	2	,,
,,	31	,,	,,	,,	28	,,	9	,,
,,	33	,,	,,	,,	14	,,	4	,,
,,	35	,,	,,	,,	8	,,	0	,,
,,	36	,,	,,	,,	37	,,	1	,,
,,	37	,,	,,	,,	16	,,	3	,,
,,	38	,,	,,	,,	12	,,	0	,,
,,	40	,,	,,	,,	28	,,	0	,,
,,	41	,,	,,	,,	27	,,	0	,,

Van Santen thus found that 70 per cent. of measles were still living after 21 days' cooling. He also abserved lively movements of the heads of some of the evaginated scolices of measles which had been chilled for 33 days. Van Santen found that measles in pieces of meat of 3 Kgm. were destroyed in three weeks, when preserved in 20 per cent. brine. He strongly advocated extending the period of chilling of measly meat to at least 40 days.

De Vries (1930) found that in his tests 17 per cent. of the measles were still capable of evaginating their scolices after 21 days' cooling.

Clarenburg (1932) described various evagination tests with measles obtained from a very heavily infested calf, which had been artificially infected. His tests were performed early in 1931. He kept his chilling-room temperature at ½° C. to 1° C., that is, just above freezing. Clarenburg found that after 3 weeks' cooling at those temperatures, putrefaction had set in, in the superficial musculature. This putrefaction had no apparent effect on the vitality of the *cysticerci*, and " even in putrefied meat very viable measles were noticeable." After 41 days' chilling he found that 12 scolices evaginated in bile solution.

Schmey and Bugge (*Berl. Tier. Woch.*, 1931), under the aegis of the German Ministry of Agriculture, did various tests and found that after 28 days' cooling *Cysticerci bovis* were still quite capable of development, and after 39-42 days definite signs of viability were noticeable, e.g. evagination of scolices, movement of the terminal organs and demonstration of "flame" cells. They, therefore, pointed out that chilling measly meat was positively dangerous, and recommended that the period be increased to at least 28 days.

In order to render the atmosphere in cooling chambers intended for prolonged chilling of measly carcasses "germ free", May (1931) recommended the modern *katadynsterilisator*, which could be prepared by painting or coating the air channels of the cooling chamber with a prepared silver solution.

In causing confirmatory tests to be applied in the United States, Mohler (1933) found that *C. bovis* was still viable after 21 days' cooling. He found that some *C. bovis* were alive in meat after 26 days' cooling, but none was alive after 31 days' cooling.

Zunker (1935) had four negative results with infectivity tests with measles taken from bovine carcasses which had been chilled for 28 days, but he was not convinced that the capability of infection was lost in measles from beef chilled for that period.

Judging from the foregoing review of recent literature on the subject, it will be concluded that the chilling of measly bovine carcasses is not safe, from the public health point of view. In the light of our present knowledge, freezing of measly carcasses would appear to be the most effective method of rendering the meat safe for human consumption, and one would go so far as to say the least damaging to meat. Von Ostertag has steadily advocated preserving measly meat in a cooler for 21 days, but apart from those writers who pointed out the risk to public health, some of his other strong arguments have been negatived by several recent workers:—

(*a*) Clarenburg, who maintained a temperature just above freezing, found that putrefaction set in comparatively early. One feels that under commercial conditions in abattoirs, very rarely will it be practicable to preserve meat for such a period before putrefaction will do damage, in spite of von Ostertag's directions regarding the maintenance of low temperatures and humidity of the air, as we have already tabulated.

(*b*) Von Ostertag's claim that under *chilling* conditions the least loss of weight will occur, has been refuted, practically, by Wagemann (1935), who staunchly preferred the freezing of measly carcasses to the uncertain 21 days' cooling method. Wagemann pointed out that carcasses frozen from four to six days, so that the internal temperature registered by spear-thermometers, reached -3° C., showed a loss in weight of 3·4 to 6 Kgm., which amounted to 2·4 per cent. to 2·43 per cent. In comparison, Wagemann showed that in the case of 21 days' cooling, the loss of weight was 11 to 15 Kgms., amounting to 4·22 per cent. to 5·88 per cent.

Wooldridge (1933) mentioned that when meat is frozen, the water within the muscle fibre separates out as ice, which is formed in the spaces between the fibres; on thawing, the separated water containing a certain amount of nutritive matter and haemoglobin, drains away. This " drip " is more copious with beef than with mutton. However, if beef is frozen sufficiently rapidly the ice is formed within the fibre and no " drip " occurs on thawing.

Keller advised that thawing should take place slowly, so as to avoid a loss of juice. According to him a temperature of 5° C. is satisfactory. After the quarters have been thawed, the temperature should be quickly reduced by strong air current to about 0° C. This should be followed by a hanging in the halls for several days at a temperature between 0° C. and 4° C. to complete the " freshness " of the meat.

Kallert also suggested slow thawing of frozen carcasses, which should take four to five days, according to the weight of the carcass, at temperatures of 5° C. to 8° C.

At least one of the earliest investigators into the subject (Glagé, 1896) pleaded that freezeing was the most effective and safest method of rendering measly pork fit for human consumption.

Reissman (1897) found that *C. bovis* died within three days when kept at temperatures of - 8° C. to -10° C. in the depths of pieces of meat. Under similar conditions *C. cellulosae* died within 4 days.

Boccalari (1903) found that both *cysticerci* died within six days at 0° C. to -2° C. (Mönnig, 1928).

Ransom (1914) used two heavily infested bovine carcasses, which, after having been kept in a cooler for 24 hours, were quartered and hung in a freezing chamber at temperatures between 11° F. and 15° F. He retained one quarter in the chilling room as a control. After six days' continuous freezing he examined 63 *cysticerci* on a warm stage and found no movement. He then swallowed six cysts and after 18 weeks was still free from tapeworm infection. On the eighth day all twelve measles from his control quarter of beef in the chilling room were still alive.

Killisch (1923) found that by freezing half-carcasses of pigs at temperatures of -8° C. to -12° C. for 3½ days all *cysticerci* were dead. He performed his tests with five heavily infested pig carcasses, which were delivered to him without skin or fat. He found that a 24-pound ham was frozen through after 48 hours, at a temperature of -8° C. to -10° C., and all measles were dead. In a 10-lb. ham, freezing was complete in the deepest muscles after 66 hours and all measles were dead after 7 hours at -6° C. to -8° C. In a 8½-lb. ham freezing at -2·5° C. to -6° C. killed all measles in 125 hours. Similarly, at that temperature all measles were killed in a 20-lb. ham in 150 hours. In a very large ham all measles were killed at -12°C. to -18°C. in 60 hours and with certainty within 72 hours at -10° C. to -15° C. He noticed that circumference of a ham which measured 64 cm. around its ~~widest~~ part was increased by 2·5 cm. by freezing at -12° C. to -18° C. for 66 hours. Working also on *C. cellulosae* Brohmann (1924) confirmed Killisch's findings.

M. Müller (1923) confirmed the experiments of Wagner (1922) and showed that after 8 days' freezing of measly bovine carcasses no measles were found to be viable. He also tested *C. cellulosae* from measly pork carcasses which arrived at his abattoir in a frozen state. Some of these *cysticerci* were still able to show slight movements, but none evaginated their scolices. Müller names this as proof of the weak resistance of *C. cellulosae* to temperatures below zero.

Van Oijen (1929) and later van der Slooten (1936) pointed out the futility of treating measly carcasses under conditions of cooling. "The only safe way is to freeze measly carcasses for at least 10 days at -10° C." (van der Slooten).

Schmey and Bugge (1930 and 1931) found that isolated measles were killed in three to four hours by freezing at -8° C. to -10° C. They found that a temperature of -6° C. was reached in three to four days in the innermost tissues of large pieces of meat at room temperatures of -8° C. to -10° C. According to those authors a freezing for 4 days is in every respect sufficient. Whatever refers to *C. bovis* is equally applicable to *C. cellulosae* in pig carcasses. Lightly infected pork need thus not be boiled, but can be made safe for human consumption in four days by freezing at low temperatures. (Compare the results of our Bloemfontein investigations in section C of this Part.)

Von Ostertag (1930) once more advocated against the freezing of measly meat, and against Schmey and Bugge's recommendations in particular, i.e. freezing measly meat for three to four days at -8° C. to -10° C. His reasons were: (1) Because after 21 days' chilling of meat, the measles evaginated their scolices, it did not follow that they were infective to man. (2) Freezing degenerated the meat, and there were not freezing chambers in all abattoirs in Germany.

Clarenburg (1932) found that in pieces of meat 6 cm. in thickness, which were placed in a freezing chamber at -8° C. to -10° C., the *cysticerci* were killed after 65 hours. He found that it took about 15 hours before the interior of the meat was cooled down to the same temperature as the air inside the chamber.

Feldforth (1934), in a series of experiments, exposed portions of beef infested with *C. bovis* to a temperature of -2° C. for varying periods of time. Viability was tested either by immersion of the cysts in a bile-saline solution at 41° C., or by swallowing the cysts in small silk bags. (Iwanizky's method.) He found that 2 days at -2° C. were lethal to cysts in meat up to half-a-pound in weight.

Scheerer (1935) found that *C. bovis* rolled up in slices of meat and exposed to a temperature of -2° C. are killed after 7 days and are no longer infective after 6 days. He concluded that slightly infested meat, frozen so that the innermost parts of the carcass remain at -2° C. for 6 days might safely be offered for sale. Zunker (1935) froze beef carcasses at an average temperature of -7° C. to -8° C. He read the internal temperatures of the meat by spear-thermometers, instruments encased in steel tubes with sharp points, which were inserted deeply into the musculature of the hind quarters. [Keller, 1935, in *Zeitschrift für Fleisch-und Milchhygiene* 45 (17), pp. 321-322, had mentioned that in freezing cysticercous meat by the new rapid process, which aimed at maintaining the meat at - 3° C. for 24 hours, it was not sufficient to control the temperature of the refrigerator. Keller mentioned that the more fat in a given carcass, the longer it would take to cool to - 3°C. in the interior. Hence it was considered by him essential to read the temperature within the carcass. Keller described the use of a pointed steel tube, which was driven into the meat and which protected the glass stem-graduated thermometer.] Zunker explained that the kinds of changes which the animal tissue undergoes during freezing depends mainly on the speed of the freezing process. The faster the cooling down takes place, the finer is the crystal conglomerate in the frozen tissue. The killing off of living tissue depends on the complete freezing out of the fluid contents of e.g. the bladderworm, and the speed with which this is done is an important factor. " Just as with special care living fish may be frozen and later again be thawed without suffering harm, so also the measles under advantageous circumstances can sometimes stand the freezing very well. During the slow freezing of the whole animal carcass, the measles are killed with certainty, however."

Zunker found that it was necessary to keep an infected carcass in the freezing chamber until the temperature in the depths of the hind quarters registered - 3° C. Even in the case of heavy carcasses, this took place within 7 days, and for safety's sake a further 24 hours could be allowed. In other words, Zunker recommended that measly carcasses should be frozen 24 hours longer than the time required for the temperatures in the deep muscles to reach - 3° C. As an example he mentioned one quarter of beef, in which an internal temperature of - 3·7° C. was reached in 5 days. That quarter, according to Zunker, was fit for issue on the 6th day. He was a staunch advocate of the freezing process of treatment for measly carcasses, in preference to the somewhat uncertain cooling process.

Kallert (1931) maintained that whereas certain plants and animals, according to Pictet, could withstand tremendous freezing, e.g. fishes frozen into blocks of ice at - 15° C. survived by careful thawing, but were killed outright at - 20° C.; frogs could withstand a temperature of - 28° C., millipedes - 50° C., snails - 120° C., *cysticerci* belonged to the creatures which were very susceptible to freezing and were definitely killed in freezing temperatures. He found that only at temperatures of - 6° C. to - 8° C. all meat juice was frozen. Kallert quotes the experiments of Kallert and Plank, who found that hindquarters weighing 60 Kgm. were completely frozen through at temperatures of - 6° to - 8° C. in 6 to 7 days, while in fore-quarters this occurred in 5 days. According to Kallert, hind-quarters should be frozen at - 8° C. to - 10° C. for at least 10 days, and fore-quarters for at least 9 days. He found that a superficial layer of fat greatly retarded cooling in a carcass, the heat conductivity of fat being 35 per cent. less than that of muscle. Large and heavy quarters required 3 per cent. longer time for complete through freezing.

Annie Porter, working in Johannesburg in 1923, " under commercial conditions ", was probably alone among all modern observers in the results she obtained. She compared the resistance of *cysticerci* to the influence of cold, with those of fishes and snails, as was mentioned by Pictet, and felt that only after extremely prolonged freezing were *cysticerci* rendered innocuous. According to Porter's staining reactions, *C. bovis* would appear normal in the deeper parts of the carcass after the 49th day of freezing, while some *C. cellulosae* appeared unchanged in the deep parts on the 156th day. Her freezing-room temperatures varied between -7·28° C. and -16·2° C. On two occasions she swallowed *C. bovis* which had been frozen for 10 weeks and 12 weeks, respectively, with negative results. She also obtained negative results by feeding fresh and frozen *Cysticerci bovis* to puppies and rats. On the other hand, she based criteria of viability of *C. bovis* and *C. cellulosae* under freezing conditions on several results she obtained from freezing *C. fasciolaris* and *Echinococcus* cysts, and thereby infecting a cat and a dog respectively. A rat liver, containing *C. fasciolaris* was frozen for 30 days, and fed to a cat, which in due course developed *T. taeniaeformis.* A dog fed on a sheep lung containing *Echinococcus* cysts, which had been frozen for a month, developed *T. echinococcus.* Porter concluded that freezing at temperatures ranging from -5° C. to -18° C. for a period of about 10 weeks appeared to destroy the vitality of all the *cysticerci* in carcasses of beef and pork. For safety, according to Porter, a margin should be allowed on this, and a period of at least 12 weeks' freezing of slightly infested beef or pork at a temperature of 14° F., that is -10° C., should be undergone before the meat may be regarded as sterile and the *cysticerci* as dead.

A word of warning was expressed by Keller (1936), who stressed the necessity of maintaining a low temperature in freezing measly carcasses. He showed that by keeping pieces of beef infected with *C. bovis* at temperatures between -1° C. and -1·5° C. not all *cysticerci* were killed even after 23 days at this temperature, but 50 per cent. were killed after 11 days. At such a temperature the meat is frozen, but the host capsule surrounding the *cysticercus* itself is not frozen. According to Keller, at such high temperatures of freezing, infectivity, tested by Iwanizky's method, is retained until at least the eleventh day.

According to Mönnig (1928), about 1914, Ottesen found a method of freezing fish in a 21 per cent. solution of sodium chloride at -10° C. to -15° C., in which the fish froze ten to twenty times more rapidly and with much less loss of weight than in air.

Plank and Kallert (1915), referred to by Kallert (1923) and by Mönnig (1928), confirmed the claims of Ottesen, but they found that with larger pieces of beef, half pig carcasses and whole sheep carcasses, the period of total freezing was shortened only about eight times. In the experiments described by Kallert, the temperature of the salt solution was -14° C. to -15° C. He pointed out that halves of pig carcasses which were frozen through in three days at -6° C. to -8° C. in the air, were frozen through in three to four hours in brine. He claimed also, that smaller pieces of beef and lighter sheep carcasses would freeze sooner in brine. He found that there was less loss of weight of carcasses frozen in brine than of those frozen in air. According to de Jong (1922), meat frozen in brine left no salty taste, and could be delivered to consumers as in the case of fresh meat.

According to Kallert a small amount of salt did penetrate into the tissues.

Drooglever Fortuyn (1922) (Mönnig, 1928), made comparative histological studies of normal meat, meat frozen in air, and meat frozen according to the above method. He found that refrigeration in air compressed the muscle fibres and drove them together in groups, relatively large cavities coming into existence between such groups; refrigeration in salt solution causes the appearance of cavities in the individual fibres but no compression of fibres.

Mönnig (1928) mentioned that the *cysticercus* is well protected by its vesicle, and he was inclined to think that the more rapid death of the *cysticerci* under this method of treatment, as was found by Brohmann (1924), was due to the very rapid freezing.

According to Brohmann, Ottesen found more rapid freezing of fish in 21 per cent. brine, if the solution was continuously stirred Brohmann did not stir the solution. He strongly recommended freezing measly pigs in brine at - 15° C. He found that by freezing pigs in brine at that low temperature all measles were destroyed in 12 hours. Unfortunately, he made no infection tests, but relied mainly on evagination tests in bile-saline solutions, for his criteria. Mönnig (1928) supplies the following summary of Brohmann's results:—

Shoulder 6 hours in brine at - 6° to - 8° C. 55/55 *cysticerci* alive.

Ham 6 hours in brine at - 10° C. 46/46 *cysticerci* alive.

Shoulder 8 hours in brine at - 5·55° C. 40/40 *cysticerci* alive.

Ham 12 hours in brine at - 10° C. to - 11° C. 42/42 *cysticerci* alive.

Shoulder 8 hours in brine at - 13° C. 40/40 *cysticerci* alive.

Ham 12 hours in brine at - 15° C. to - 16·5° C. 0/48 *cysticerci* alive.

" The pieces were all completely frozen when removed from the brine. *Cysticerci* from all depths were examined after the pieces had been allowed to thaw at room-temperature." (Mönnig.)

Schmey and Bugge (1930) pointed out that refrigeration technique had advanced so far that refrigeration in brine would result in obtaining a temperature of - 6° C. in a few hours.

C. Viability Tests with Measles taken from Chilled and Frozen Pork and Beef Carcasses at the Bloemfontein Abattoir.

The object of these tests was mainly to confirm the tests of overseas writers, and, if possible, to establish definitely, with what material we had available, the shortest period required for the freezing of measly beef and pork at temperatures of approximately - 10° C., under South African commercial conditions.

METHOD EMPLOYED.

(1) After the stipulated period of chilling or freezing, the *cysticerci* (*cellulosae* or *bovis*) were always removed from their connective tissue capsule.

(2) Physical characteristics of such treated *cysticerci* were observed, although no microscopic observations of loosening of the hooks in *C. cellulosae* were made.

(3) Control evagination tests in bile-saline solution and in sodium taurocholate were made in an incubator at 38° C.

(4) The main criteria in these tests were based on actual infection tests on several voluntary assistants, according to both Iwanizky's and Keller's methods.

(5) In view of the fact that the infection tests perfected by Iwanizky and by Keller have very nearly approached conditions of natural infection, it was not considered necessary to attempt reactions to warming on a stage, and the observations of the physical characteristics of frozen or chilled measles were merely those of noting cloudiness, discolouration and consistency of the fluid.

(6) Since the boiling or pickling of measly carcasses is not practised in South Africa, such tests were not attempted. All available material was, therefore, used in cooling and freezing tests.

With regard to control artificial evagination tests, we found the best results with 5 per cent. sodium taurocholate solution (Malkani's method). The *cysticerci* were carefully isolated from their connective tissue capsules and placed in a saucer containing 5 per cent. sodium taurocholate solution, which was then placed in an ordinary " Buck-Eye " egg incubator, of which the heat was regulated to 38° C. Evagination of scolices usually occurred within two hours in the case of viable *cysticerci*. It was found that this method worked very successfully with fresh (unfrozen) *cysticerci*, more sluggishly with those frozen for about 24 to 48 hours, or chilled for about 21 days, and frequently did not work with those measles from meat chilled longer than about 21 days, or frozen longer than 48 hours. On the other hand, we established definitely that the only sure criterion of viability of measles could be obtained by actual infection tests according to Iwanizky's and/or Keller's methods. In some cases we failed to obtain evagination *in vitro* of scolices of measles from meat frozen for three days and more, whereas ready and clear evaginations of such scolices occurred within the bag or tube in our subject's intestine. We used a slight modification of both Iwanizky's and Keller's methods. In the former we found that it frequently took some time to stitch the bags, as Iwanizky did, and frequently the sutures did not appear too secure. In order, therefore, to instil the fullest confidence in his personal safety in our subject, we tied the small silk bags with strong suturing silk, and cut the tied end as short as possible. All our subjects, European members of the Abattoir Staff, had no difficulty in swallowing them, and one subject in particular, never failed to recover the bags, or the celluloid tubes in his stools. With reference to our modification of Keller's method, we found the same objection to suturing the silk cover. A single layer of silk was, therefore, wrapped round the celluloid tube containing from one to three measles, in such a way that the two open ends of the small cylinder were covered with a single layer of silk, drawn tautly, and the four corners of the silk coverlet were twisted together in a spiral and tied as closely to the body of the cylinder as possible. This spiral twisting caused the two single silk-layers covering the ends of the tubes to be drawn even more tightly, almost like a drum, than in the case of suturing the coverlet, or else fixing by means of artificial fixatives, e.g. acetone-cellulose solution, which took a considerable time to fix, and was treated with a certain amount of suspicion by our " chief " subject. Most of our subjects experienced a measure of discomfort in swallowing the hard celluloid tubes, measuring approximately 15 mm. long by 7 mm. diameter. Our " chief " subject, however, once more found no difficulty in the deglutition of the somewhat unwieldy " pills ", and for that reason experiments by means of Keller's method were confined to him, since he was quite prepared to use the celluloid tubes over and over again, he never " lost " any, and I was only able to obtain the tubes through the kind favour of the Director of Veterinary Services, Onderstepoort Laboratory, and could therefore, not abuse his favour by repeatedly applying for fresh tubes. The inner temperature of the meat in the freezing chamber was read on improvised spear-thermometers, ordinary low graded freezing thermometers, encased in sharp-pointed steel covers. One of these was kindly made for me by the Mechanical Engineer in charge of my Abattoir Refrigeration plant, and two were lent by the Director of Veterinary Services, Onderstepoort. (See Table A.)

Evagination tests with *C. cellulosae* from pork cooled for various periods have thus shown that the most successful results have been obtained with natural infection tests, according to Iwanizky's and/or Keller's methods.

In five per cent. sodium taurocholate, evagination nearly always occurred within two hours, and by using 30 per cent. pig bile-physiological saline solution in from 2 to 5 hours. After that, evaporation of the fluid contents of the saucer often occurred. In fresh measles, namely those which had not undergone prolonged chilling or freezing, evagination occurred more readily, and in a shorter time. After about the twenty-first day of chilling, putrefaction frequently set in, in the pork, but, according to the results obtained by our natural infection tests, it did not follow that the measles situated in badly putrefied areas, died in that situation. By our tests we established the fact that the *Cysticercus cellulosae* can remain infective up to 41 days after slaughter of the host, but we failed to find any alive after that day, although we only tried two more tests, on account of the undesirability of maintaining putrefied meat in the condemned meat section of our chilling room.

It is interesting to mention that after 35 to 41 days' chilling, we found only 4 out of 167 tested measles viable. After 34 days' chilling, 12 out of 30 pig measles were still viable; and after 30 to 33 days' chilling, 43 out of 123 pig measles tested were still viable.

TABLE A.

Evagination Tests with C. cellulosae—*Chilling Tests.*

Experiment No.	Part of carcass (weight).	Number of days chilled.	Method used: I. Iwanizky, K. Keller.	Number of Human subjects.	Number of tubes or bags swallowed.	Number recovered.	Total number of cysts swallowed.	Number of scolices Evaginated.	Number of cysts digested (dead).	Remarks.
1	Shoulder (21 lb.)	4	I.	3	10 B.	9	10	6	3	One bag was lost; 6/10 scolices evaginated in 5 % sod. taurocholate sol.
2	Shoulder (22 lb.)	6	I.	2	4 B.	4	8	6	2	4/10 Scolices evaginated in 5 % sod. taurochol.
3	Leg (18 lb.)	1	K.	1	4 T.	4	8	8	0	10/10 Scolices evaginated in 2 hours in 5 % sod. taurochol. and 7/10 in 30 % pig bile.
4	Leg (18 lb.)	2	K.	1	4 T.	4	8	7	1	4/10 Scolices evaginated in 30 % pig bile.
5	Leg (27 lb.)	3	K.	1	3 T.	3	9	9	0	—
6	Leg (30 lb.)	4	K.	1	3 T.	3	6	6	0	6/10 Scolices evaginated in 5 % sod. taurochol. and 4/10 in 30 % pig bile.
7	Leg	5	K.	1	2 T.	2	6	6	0	—
8	Shoulder (24 lb.)	6	K. I.	2	2 T. 2 B.	4	12	10	2	5/10 Scolices evaginated in 5 % sod. taurochol. and 2/10 in 30 % pig bile.
9	Shoulder (24 lb.)	7	K. I.	2	2 T. 3 B.	5	14	13	1	3/7 Scolices evaginated in 5 % sod. taurochol. and 1/7 in 30 % pig bile.
10	Shoulder (21 lb.)	8	K. I.	2	3 T. 3 B.	5	17	14	0	3 Cysts in 1 bag lost.
11	Leg (—)	9	K. I.	1	3 T. 3 B.	6	16	16	0	2/5 Scolices evaginated in 5 % sod. taurochol. and 1/6 in 30 % bile.
12	Leg (—)	11	K. I.	1	3 T. 3 B.	6	16	16	0	— —
13	Shoulder (—)	12	K. I.	1	3 T. 3 B.	6	12	9	3	—
14	Leg (17 lb.)	13	I.	1	5 B.	5	15	0	15	Meat was badly putrefied, and measles were stirkily opaque. All artificial tests failed.

TABLE A—(*continued*).

Experiment No.	Part of carcass (weight).	Number of days chilled.	Method used : I. Iwanizky. K. Keller.	Number of Human subjects.	Number of tubes or bags swallowed.	Number recovered.	Total number of cysts swallowed.	Number of scolices Evaginated.	Number of cysts digested (dead).	Remarks.
15	Leg (32 lb.)	14	K. I.	1	3 T. 3 B.	6	18	10	8	—
16	Leg (30 lb.)	15	K. I.	1	2 T. 2 B.	4	10	10	0	5/6 Scolices evaginated in 5 % sod. taurochol. and 2/7 in 30 % pig bile.
17	Shoulder (17 lb.)	16	K. I.	1	2 T. 2 B.	4	10	10	0	--
18	Neck (18 lb.)	18	I.	1	2 B.	2	4	3	1	–
19	Leg (21 lb.)	17	K.	1	5 T.	5	15	7	8	
20	Leg (27 lb.)	18	K.	1	4 T.	4	12	9	3	3/6 scolices evagin. in 5 per cent. taurochol. and 0/5 in 30 per cent. pig bile.
21	Shoulder (17 lb.)	19	K. I.	2	6 T. 4 B.	10	30	29	1	
22	Shoulder (19 lb.)	20	K.	1	5 T.	5	15	13	2	
23	Leg (36 lb.)	21	K.	2	4 T.	6	20	15	2	One bag with 3 cysts lost ; 3/10 scolices evagin. in 5 per cent. Sod. taurochol. and 2/10 in 30 per cent. pig bile.
24	Part of Leg	22	K.	1	4 T.	4	12	8	4	
25	Leg (31 lb.)	23	K.	1	4 T.	4	10	10	0	
26	Part of Leg	24	K.	1	4 T.	4	10	10	0	
27 & 28	Leg and Shoulder	25	K.	1	3 T. 5 T.	8	25	6 13	4 2	Two experiments on subsequent days.
29 & 34	Shoulder and Shoulder	26	K.	1	5 T. 4 T.	9	25	12 0	3 10	Two experiments with 10 days interval. Pork from different carcasses.
30 & 35	Leg Leg	27	K.	1	5 T. 5 T.	10	30	12 3	3 12	Two experiments and pork from same respective carcasses as 29 and 34.
31 & 36	Shoulder Leg	28	K. I.	2	3 T. 2 B. 5 T. 3 B.	13	39	7 9	8 15	Two experiments, pork from different carcasses.

TABLE A—(*continued*).

Experiment No.	Part of carcass (weight).	Number of days chilled.	Method used : I. Iwanizky. K. Keller.	Number of Human subjects.	Number of tubes or bags swallowed.	Number recovered.	Total number of cysts swallowed.	Number of scolices Evaginated.	Number of cysts digested (dead).	Remarks.
32 & 37	Leg Shoulder	29	K. I.	2	3 T. 2 B. 4 T. 4 B.	13	35	5 7	10 13	Two experiments, pork from same carcasses as 31 and 36.
33 & 38	Leg Leg	30	K. I.	3	4 T 2 B. 3 T. 2 B.	11	32	7 0	10 15	Two experiments with different carcasses.
39	Shoulder	31	K. I.	2	5 T. 5 B.	8	30	20	4	Two bags with 6 cysts lost.
40	Leg	32	K. I.	2	5 T. 5 B.	10	31	13	18	No artificial tests tried with experiments 24 to 41 inclusive.
41	Leg	33	K. I.	1	5 T. 5 B.	9	30	3	24	One bag with 3 cysts lost.
42	Shoulder	34	K. I.	1	5 T. 5 B.	10	30	12	18	0/10 scolices evagin. in 5 per cent. taurochol. and 0/9 in 30 per cent. pig bile.
43	Shoulder	35	K. I.	1	5 T. 5 B.	10	30	1	29	0/10 scolices evagin. in 5 per cent. taurochol. and 0/9 in 30 per cent. pig bile.
44	Leg	36	K. I.	1	5 T. 3 B.	8	24	0	24	0/10 scolices evagin. in 5 per cent. taurochol. and 0/9 in 30 per cent. pig bile.
45	Leg	37	K. I.	1	4 T. 4 B.	8	24	2	22	Two scolices in 2 tubes evaginated ; none in the bags.
46	Carcass	38	K. I.	1	4 T. 4 B.	8	20	0	20	
47	Carcass	39	K. I.	1	4 T. 1 B.	5	14	0	14	
48	Carcass	40	K. I.	1	5 T. 6 B.	8	40	0	32	Three bags lost with 8 cysts.
49	Leg	41	K. I.	1	3 T. 2 B.	5	15	1	14	
50	Leg	42	K. I.	1	3 T. 2 B.	5	15	0	15	
51	Shoulder	50	K.	1	3 T.	3	9	0	9	Meat badly putrefied.

For illustrations see Figures 4 to 7.

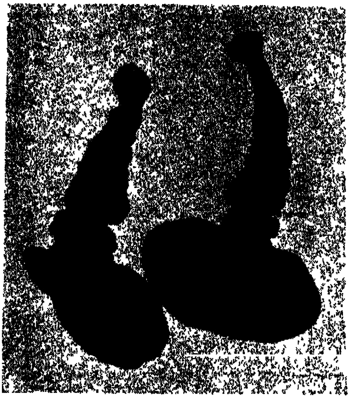

FIGURE 4.

C. cellulosae scolices evaginated in 2 hours in 30 per cent. pig bile-saline solution. (2 days chilled.) Magnification 7 times.

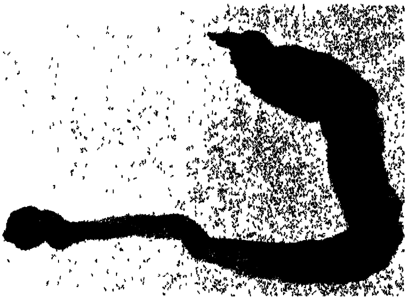

FIGURE 5.

C. cellulosae scolices evaginated by Keller's method, after 19 days' cooling. Magnification 7 times.

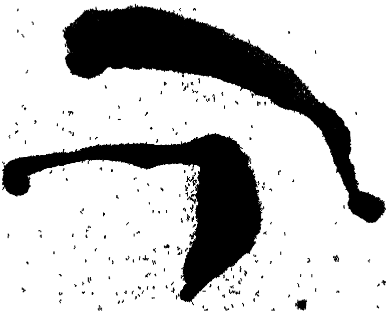

FIGURE 6A.

C. cellulosae scolices evaginated Keller's method after 28 days' cooling. Magnification 7 times.

FIGURE 6B.

Microscopic view of head of same, showing suckers and rostellum. Magnification 40 times.

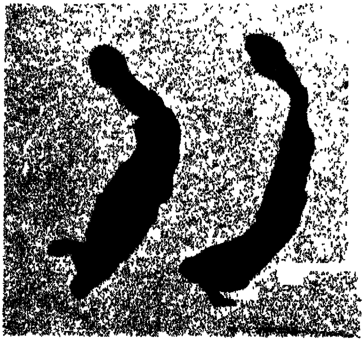

FIGURE 7.

C. cellulosae scolices, evaginated Keller's method after 32 days' chilling. Magnification 7 times.

Freezing Tests with Cysticercus cellulosae.

Experiment No.	Number of Days in Freezer.	Temp. of Meat after 24 Hours Cooling (°F.).	Initial Temp. of Freezing Chamber (°F.).	Temp. of Freezing Chamber after 5 Hours (°F.).	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 24 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 48 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 3 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 4 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 5 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 6 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 7 Days.	Part of Carcass. (Weight.)	Method Iwanizky (I.). Keller (K.).	Number of Cysts Swallowed.	Scolices Evaginated.	Digested (Dead).	Remarks.
1	14	—	—	—	—	—	—	—	—	—	—	Carcass	—	35	—	35	1
2	12	—	—	—	—	—	—	—	—	—	—	Carcass	—	25	—	25	2
3	6	—	13	—	(1) — (2) 14	(1) — (2) 13	(1) — (2) 15	(1) — (2) 14	(1) — (2) 12	— (2) 13	—	Leg (30 lb.)	I.	12	—	12	8
4	4	—	12	—	(1) — (2) 13	(1) — (2) 13	(1) — (2) 12	(1) — (2) 16	—	—	—	Shoulder	K.	4	—	4	6
5	1	—	15	—	(1) — (2) 14	—	—	—	—	—	—	Leg (24 lb.)	K.	6	6	—	3
6	2	—	14	14	(1) 19 (2) 13	(1) 16 (2) 14	—	—	—	—	—	Leg (26 lb.)	K.	6	3	3	4
7	3	—	17	15	(1) 18 (2) 12	(1) 18 (2) 14	(1) 17 (2) 15	—	—	—	—	Shoulder (19 lb.)	K.	10	7	3	5
8	4	40	16	16	(1) 19 (2) 15	(1) 17 (2) 16	(1) 17 (2) 13	(1) 16 (2) 14	—	—	—	Shoulder (15 lb.)	K.	12	1	11	6
9	5	40	16	16	(1) 19 (2) 15	(1) 17 (2) 16	(1) 17 (2) 13	(1) 16 (2) 14	(1) 16 (2) 15	—	—	Shoulder (16 lb.)	K. I.	8	8	8	7
10	6	40	16	16	(1) 19 (2) 15	(1) 17 (2) 16	(1) 16 (2) 13	(1) 16 (2) 14	(1) 16 (2) 15	(1) 15 (2) 15	—	Leg (33 lb.)	K. I.	18	—	18	8
11	7	40	16	16	(1) 19 (2) 15	(1) 17 (2) 16	(1) 17 (2) 13	(1) 16 (2) 14	(1) 16 (2) 15	(1) 15 (2) 15	(1) 16 (2) 18	Leg (38 lb.)	K. I.	13	—	13	9
12	4	41	18	19	(1) 18 (2) 15	(1) 18 (2) 13	(1) 16 (2) 11	(1) 16 (2) 13	— —	— —	— —	Shoulder (16 lb.)	I.	18	—	18	6
13	5	42	18	19	(1) 18 (2) 15	(1) 18 (2) 13	(1) 16 (2) 11	(1) 16 (2) 13	(1) 18 (2) 14	— —	— —	Shoulder (19 lb.)	K. I.	18	—	18	7

TABLE B—*(continued)*.

Experiment No.	Number of Days in Freezer.	Temp. of Meat after 24 Hours Cooling (°F.).	Initial Temp. of Freezing Chamber (°F.).	Temp. of Freezing Chamber after 5 Hours (°F.).	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 24 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 48 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 3 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 4 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 5 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 6 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 7 Days.	Part of Carcass. (Weight.)	Method Iwanizky (I.). Keller (K.).	Number of Cysts Swallowed.	Scolices Evaginated.	Digested (Dead).	Remarks.
14	1	39	15	15	(1) 20 (2) 16	—	—	—	—	—	—	Leg (21 ℔.)	K. I.	15	5	10	3
15	2	39	15	15	(1) 20 (2) 16	(1) 18 (2) 15	—	—	—	—	—	Leg (19 ℔.)	K. I.	15	—	15	4
16	3	39	15	15	(1) 20 (2) 16	(1) 18 (2) 15	(1) 18 (2) 15	— —	— —	— —	— —	Shoulder (18 ℔.)	K.	10	—	10	5
17	4	39	15	15	(1) 20 (2) 16	(1) 18 (2) 15	(1) 18 (2) 15	(1) 17 (2) 15	— —	— —	— —	Shoulder (19 ℔.)	K. I.	15	—	15	6
18	1	41	19	19	(1) 23 (2) 16	—	—	—	—	—	—	Leg (42 ℔.)	K.	7	4	3	3
19	2	41	19	19	(1) 23 (2) 16	(1) 20 (2) 15	—	—	—	—	—	Leg (44 ℔.)	K.	8	—	8	4
20	3	41	19	19	(1) 23 (2) 16	(1) 20 (2) 15	(1) 18 (2) 16	—	—	—	—	Leg (26 ℔.)	K.	8	—	8	5
21	3	40	20	20	(1) 23 (2) 17	(1) 20 (2) 16	(1) 18 (2) 15	—	—	—	—	Carcass (185 ℔.)	K. I.	60	—	60	5
22	4	36	14	16	(1) 21 (2) 19	(1) 21 (2) 13	(1) 18 (2) 13	(1) 18 (2) 13	—	—	—	Carcass (87 ℔.)	K. I.	40	—	40	6
23	4	38	16	16	(1) 18 (2) 14	(1) 17 (2) 14	(1) 17 (2) 11	(1) 15 (2) 11	— —	— —	— —	Carcass (105 ℔.)	K. I.	36	—	36	6
24	5	40	13	14	(1) 19 (2) 16	(1) 18 (2) 14	(1) 18 (2) 15	(1) 16 (2) 13	(1) 17 (2) 17	— —	— —	Carcass (71 ℔.)	K. I.	50	—	50	7
25	5	41	15	16	(1) 23 (2) 16	(1) 20 (2) 14	(1) 17 (2) 15	(1) 17 (2) 16	(1) 15 (2) 9	— —	— —	Carcass (161 ℔.)	K. I.	40	—	40	7
26	5	38	12	13	(1) 20 (2) 11	(1) 16 (2) 13	(1) 16 (2) 14	(1) 15 (2) 11	(1) 15 (2) 12	—	—	Carcass (48 ℔.)	K. I.	30	0	30	7

TABLE B—(*continued*).

Experiment No.	Number of Days in Freezer.	Temp. of Meat after 24 Hours Cooling (°F.).	Initial Temp. of Freezing Chamber (°F.).	Temp. of Freezing Chamber after 5 Hours (°F.).	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 24 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 48 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 3 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 4 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 5 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 6 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 7 Days.	Part of Carcass. (Weight.)	Method Iwanizky (I.). Keller (K.).	Number of Cysts Swallowed.	Scolices Evaginated.	Digested (Dead).	Remarks.
27	5	39	18	16	(1) 18 (2) 14	(1) 18 (2) 15	(1) 17 (2) 13	(1) 14 (2) 12	(1) 15 (2) 13	— —	— —	Carcass (61 lb.)	K. I.	40	—	40	7
28	5	40	16	15	(1) 17 (2) 15	(1) 16 (2) 14	(1) 16 (2) 14	(1) 15 (2) 13	(1) 14 (2) 13	— —	— —	Carcass (86 lb.)	K. I.	30	—	30	7
29	5	39	16	16	(1) 21 (2) 14	(1) 20 (2) 15	(1) 19 (2) 12	(1) 18 (2) 10	(1) 17 (2) 13	— —	— —	Carcass (101 lb.)	K. I.	30	—	30	7
30	5	41	14	13	(1) 20 (2) 14	(1) 18 (2) 13	(1) 17 (2) 13	(1) 17 (2) 15	(1) 17 (2) 17	— —	— —	Carcass (62 lb.)	K. I.	60	—	60	7
31	5	39	13	14	(1) 18 (2) 13	(1) 16 (2) 12	(1) 16 (2) 14	(1) 15 (2) 13	(1) 15 (2) 15	—	—	Carcass (81 lb.)	K. I.	35	—	35	7
32	5	42	14	14	(1) 22 (2) 15	(1) 22 (2) 13	(1) 21 (2) 14	(1) 20 (2) 16	(1) 16 (2) 12	— —	— —	Carcass (202 lb.)	K. I.	20	—	20	7
33	5	37	18	17	(1) 19 (2) 17	(1) 18 (2) 15	(1) 18 (2) 17	(1) 18 (2) 12	(1) 17 (2) 14	— —	— —	Carcass (72 lb.)	K. I.	30	—	30	7
34	5	38	17	17	(1) 21 (2) 17	(1) 21 (2) 16	(1) 21 (2) 14	(1) 20 (2) 15	(1) 19 (2) 13	—	—	Carcass (98 lb.)	K. I.	30	—	30	7
35	5	39	16	18	(1) 20 (2) 16	(1) 21 (2) 15	(1) 21 (2) 17	(1) 18 (2) 14	(1) 18 (2) 12	— —	— —	Carcass (49 lb.)	K. I.	30	—	30	7
36	5	40	15	16	(1) 25 (2) 16	(1) 23 (2) 14	(1) 23 (2) 12	(1) 19 (2) 13	(1) 19 (2) 14	—	—	Carcass (131 lb.)	K. I.	24	—	24	7
37	5	41	11	12	(1) 20 (2) 13	(1) 19 (2) 14	(1) 19 (2) 13	(1) 19 (2) 17	(1) 19 (2) 18	— —	— —	Carcass (111 lb.)	K. I.	30	—	30	7
38	5	40	18	14	(1) 22 (2) 14	(1) 17 (2) 16	(1) 17 (2) 14	(1) 17 (2) 10	(1) 17 (2) 12	— —	— —	Carcass (36 lb.)	K. I.	30	—	30	7

Remarks Index to Foregoing Table (B).

1. A heavily infested pig carcass was frozen for 14 days. The weight of the carcass was not recorded at the time. The carcass was literally frozen to resemble a wooden box in consistency. Some of the *cysticerci* were removed and resembled crystals of ice. The carcass was thawed for 24 hours and 35 measles were removed from the innermost muscles. These were swallowed in their naked state by five members of the Abattoir Staff, N.F.V.; H.M.D.; M.C.; P.J.K.; and W.H.G.; each of whom swallowed seven measles. After twelve months, none of the subjects has developed tapeworm infection.

2. A heavily infested pig carcass was frozen for 12 days. Its physical condition resembled that of Experiment 1, after freezing. The carcass was thawed for 24 hours and 25 measles were removed from the deeper tissues and swallowed in their naked state by two natives on the staff and one European, R.P. After approximately 12 months, no tapeworm infestation has resulted.

3. *Experiment No.* 5.

Leg of pork weighing 24 lb. was frozen for 24 hours. The leg was practically frozen through. Six *cysticerci* were swallowed by W.H.G. according to Keller's method, and six scolices evaginated.

Experiment No. 14.

Leg of pork weighing 21 lb. was frozen for 24 hours. Fifteen cysts were swallowed by W.H.G. in 2 tubes and 3 silk bags. All were recovered. Five scolices (2 in each tube and 1 in 1 bag) had evaginated, and 10 presumably dead *cysticerci* were completely digested.

Experiment No. 18.

Leg of pork weighing 42 lb. was frozen for 24 hours. This was a fairly fat animal. Seven *cysticerci* in two tubes were swallowed by W.H.G. Both tubes were recovered. One contained 3 evaginated scolices and the other, one. Three *cysticerci*, presumably dead, were digested.

Contemporary artificial evagination tests were made, with the following results:—

2 out of 6 scolices evaginated in 5 per cent. sodium taurocholate solution.

1 out of 5 scolices evaginated in 30 per cent. pig bile-physiological saline solution.

Summary.

On three occasions W.H.G. swallowed a total of 28 *C. cellulosae*, which had been removed from pork frozen for 24 hours. Out of this number 15 scolices evaginated.

4. *Experiment No.* 6.

Leg of pork weighing 26 lb. was frozen for 48 hours. Meat was nearly frozen through. After 24 hours' thawing of the meat, W.H.G. swallowed two celluloid tubes, each with three measles. A total of three out of the six measles evaginated scolices. (This leg of pork was from the same carcass as that of Experiment No. 5, above.)

Experiment No. 15.

Leg of pork, 19 lb., was frozen for 48 hours. Freezing, in this case, was definitely complete. After 24 hours' thawing, 15 measles were removed and given to W.H.G. to swallow in 3 tubes and 2 bags. No scolices evaginated, and all the measles, presumably dead, were digested totally. (This leg of pork was from the same carcass as that of Experiment No. 14, above.)

Experiment No. 19.

Leg of pork weighing 44 lb. was frozen for 48 hours. Fat leg. Leg was nearly frozen through. After 24 hours' thawing of the meat, W.H.G. swallowed 8 measles in three tubes. All were digested. Artificial evagination tests in 5 per cent. solution taurocholate and 30 per cent. bile-saline solution were equally negative. This leg of pork was from the same carcass as that of Experiment No. 18.

Summary.

On three occasions W.H.G. swallowed 29 measles from pork frozen for 48 hours. Of this number, only 3 scolices evaginated.

5. *Experiment No.* 7.

A shoulder of pork weighing 19 lb. was frozen for three days. The shoulder was completely frozen through, and many of the measles resembled ice crystals. After 24 hours thawing of the meat, W.H.G. swallowed 10 measles in three tubes. The cysts were removed from the deeper part of the subscapular muscle. Of this number, 7 scolices evaginated, and three cysts were digested. The other shoulder and both hind legs were used in cooling tests. (See chilling tests Nos. 15 to 17, which showed fairly regular evagination results.)

Contemporary artificial evagination tests revealed the following negative results: 0/6 scolices evaginated in 5 per cent. taurocholate solution; 0/7 scolices evaginated in 30 per cent. pig bile-saline solution.

Experiment No. 16.

Shoulder of pork weighing 18 lb. was frozen for 3 days. Freezing was complete. After 24 hours' thawing of meat, W.H.G. swallowed 10 measles in 3 tubes. All were digested.

Experiment No. 20.

Leg of pork weighing 26 lb. was frozen for 3 days. Freezing was complete. After 24 hours' thawing of the meat, W.H.G. swallowed 8 measles. All were digested.

Experiment No. 21.

A whole carcass weighing 185 lb. was frozen for 3 days. Pig was of the large-boned, heavy variety, but not very fat. Freezing was quite complete. After 24 hours' thawing of meat, 60 measles in 5 tubes and 15 bags were swallowed by W.H.G. and three others. Two of the assistants " lost their bags. No scolices evaginated.

Summary.

On 4 occasions W.H.G. (assisted by three other persons on the last occasion) swallowed a total of 88 measles from pork frozen for three days. Of this number, 7 scolices (all on the first occasion) evaginated.

6. *Experiment No.* 4.

Shoulder of pork weighing 18 lb. was frozen for 4 days. Freezing was complete. After 24 hours' thawing of the meat, W.H.G. swallowed 4 cysts in 2 tubes. All measles were digested.

Experiment No. 8.

Shoulder of pork weighing 15 lb. was frozen for 4 days. Freezing was complete. After 24 hours' thawing of the meat, W.H.G. swallowed 12 measles in 4 tubes. Of this number, 1 scolex evaginated.

Experiment No. 12.

Shoulder of pork weighing 16 lb. was frozen for 4 days. Freezing was complete. After 24 hours' thawing of the meat, W.H.G. swallowed 18 cysts in 6 silk bags. All measles were totally digested.

Experiment No. 17.

Shoulder of pork weighing 19 lb. was frozen for 4 days. Freezing was complete. After 24 hours' thawing of the meat, W.H.G. swallowed 15 measles in 3 tubes and 2 bags. All measles were totally digested.

Experiment No. 22.

Whole carcass weighing 87 lb. was frozen for 4 days. Freezing was complete. After 24 hours' thawing, W. H. G. and P. J. K. swallowed 40 measles in 5 tubes and 8 bags. All receptacles were recovered. All measles were digested.

Experiment No. 23.

Whole carcass weighing 106 lb. frozen. W.H.G. and P.J.K. swallowed 36 measles. All measles were totally digested.

Summary.

Out of a total of 125 measles obtained from pork frozen for four days, and swallowed by W.H.G. and an assistant, only one scolex evaginated.

7. *Experiment No.* 9.

A shoulder of pork weighing 16 lb. was frozen for 5 days. Out of 8 measles swallowed, all were digested.

Experiments Nos. 24 *to* 38.

Fifteen whole carcasses of pork, weighing from 36 lb. to 202 lb., were frozen for 5 days. The heaviest carcasses, 161 lb. and 202 lb., were very fat. In each case the carcass was frozen right through. From these fifteen carcasses a total of 509 measles were swallowed by W.H.G. and three others. Of this number no scolices evaginated. A few silk bags were " lost ", but all measles in those recovered showed complete digestion. From four of the various carcasses contemporary experiments were tried in 5 per cent. sodium taurocholate solution and in 30 per cent. pig bile-physiological saline solution at 38° C. incubator temperatures, but no scolices evaginated.

Summary.

Out of 535 measles swallowed by my assistants, no scolices developed. The measles had been recovered from pork frozen for 5 days.

8. and 9. *Experiments* 3, 10 *and* 11.

Three legs of pork, each weighing approximately 30 lb., were frozen—2 for 6 days, and 1 for 7 days. Out of 43 measles swallowed, none evaginated their scolices.

The following short table summarizes the results of our freezing tests with *Cysticercus cellulosae*.

Days Frozen.	Total Measles Swallowed.	Total Evaginated.	Digested or Lost.	Percentage Viable.
1	28	15	13	53·57
2	29	3	26	10·35
3	88	7	81	7·95
4	125	1	124	0·80
5	535	0	535	–
6	30	0	30	—
7	13	0	13	—
12	25	0	25	—
14	35	0	35	—

Conclusions.

From our experiments with the freezing of measly pork, it will be noticed that at freezing room temperatures ranging from 14° F. to 19° F. (– 10° C. to – 7° C. approximately) and an internal temperature of the deeper tissues of a leg of pork of 20° F. to 23° F. (– 7° C. to – 5° C.), after 24 hours' freezing, *Cysticercus cellulosae* is still viable.

The percentage of viable measles found steadily diminishes from 2 days' freezing to 4 days' freezing, until after 5 days' freezing no more viable *Cysticerci cellulosae* were found. In view of the fact that one viable measle was found after 4 days' freezing, it would be dangerous to recommend that pork frozen for 4 days at temperatures oscillating between 13° F. and 16° F. (- 11° C. and - 9° C.) would be safe.

According to Schmey and Bugge, four days' freezing is sufficient to kill all *Cysticerci cellulosae*, and according to Killisch, all *C. cellulosae* are destroyed in half-pig carcasses in 3½ days. At Bloemfontein, on the other hand, we found that from a light shoulder of pork, weighing only 15 lb., and frozen for four days, 1 out of 12 measles was still viable. It is interesting to record that a temperature of 19° F. (- 7° C.) was reached in the depths of the subscapular muscle within 24 hours, and of 16° F. (- 10° C.) within 4 days.

From 17 experiments performed with measles from pork frozen for five days (including 15 whole carcasses), our results justify the presumption that even in heavy and fat (202 lb.) pig carcasses, *Cysticerci cellulosae* are destroyed. In the 202 lb. pig carcass (Experiment No. 32) the inner temperature registered by means of a steel-pointed thermometer inserted deeply into the musculature of the hind leg, reached 22° F. (- 5·5° C.) in 24 hours, and 16° F. in 5 days. During that period the freezing room temperature oscillated between 12° F. and 16° F.

It is presumed that very few pig measles will remain viable if subjected to continuous freezing at - 10° C. in pork, for 5 days. A safety margin of 2 days can be allowed, and after 7 days' freezing, lightly infested pig carcasses, provided they are not too fat, can safely be passed as fit for human consumption. There can be absolutely no objection, from a public health point of view, to the treatment of lightly infested measly pork carcasses, no matter how fat they are, for fourteen days at - 10° C. continuous freezing, as South African Meat Regulations provide for at present, although few abattoirs make use of the concession.

CHILLING TESTS WITH CYSTICERCUS BOVIS.

Only four such tests were performed at Bloemfontein, and one ox carcass was quartered, the quarters being kept in the cooler for 20 days, 27 days, 30 days and 31 days. Viability of the *cysticerci*, according to Keller's method, was tested, and scolices evaginated to the extent of 6 out of 14, from a fore quarter chilled for 20 days. No scolices evaginated from 17 measles from a hind-quarter chilled for 27 days. Putrefaction had, by then set in. At 30 days, the remaining fore-quarter and hind-quarter had badly putrefied, but, nevertheless, ten measles were removed from each after 30 days' and 31 days' chilling, respectively. These were also tested according to Keller's method, but no evagination of scolices occurred.

Since South African Regulations do not prescribe a period of 21 days' chilling, or longer, as an alternative to the freezing method of rendering slightly infested measly beef fit for human consumption, I

TABLE C.

Freezing Tests with Cysticerous bovis.

Experiment No.	Number of Days in Freezer.	Temp. of Meat after 24 Hours Cooling (°F.).	Initial Temp. of Freezing Chamber (°F.).	Temp. of Freezing Chamber after 5 Hours (°F.).	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 24 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 48 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 3 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 4 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 5 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 6 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 7 Days.	Part of Carcass. Weight (lb.)	Method used— I.—Iwanizky. K.—Keller.	Number of Cysts Swallowed.	Number of Cysts Evaginated.	Number of Cysts Digested (Dead).	Remarks Index.
1	1	44	19	—	(1) 29 (2) 14	—	—	—	—	—	—	Side of beef (248 lb.)	K.	4	4	0	1
2	2	41	17	—	(1) 28 (2) 15	(1) 27 (2) 13	—	—	—	—	—	Side of beef (262 lb.)	K.	2	0	2	2
3	2	—	16	—	(1) — (2) 17	(1) — (2) 12	—	—	—	—	—	Ox tongue	K.	8	0	8	2
4	1	44	20	—	(1) 23 (2) 9	—	—	—	—	—	—	Side of beef (361 lb.)	K.	10	10	0	1
5	3	44	20	—	(1) 22 (2) 9	(1) 22 (2) 11	(1) 20 (2) 10	—	—	—	—	Side of beef (350 lb.)	K.	14	7	7	3
6	2	41	12	—	(1) 24 (2) 11	(1) 23 (2) 11	—	—	—	—	—	Side of beef (254 lb.)	K.	12	1	11	2
7	1	39	12	—	(1) 23 (2) 15	—	—	—	—	—	—	Side of beef (245 lb.)	K.	8	8	0	1
8	3	41	14	—	(1) 25 (2) 16	(1) 23 (2) 14	(1) 23 (2) 11	—	—	—	—	Side of beef (250 lb.)	K.	12	9	3	3
9	3	42	14	—	(1) 28 (2) 18	(1) 24 (2) 12	(1) 23 (2) 11	— —	— —	— —	— —	Side of beef (248 lb.)	K.	10	2	8	3
10	4	43	19	14	(1) 28 (2) 10	(1) 22 (2) 12	(1) 23 (2) 19	(1) 14 (2) 14	— —	— —	— —	Side of beef (300 lb,)	K.	10	4	6	4

TABLE C—(*cont*

Experiment No.	Number of Days in Freezer.	Temp. of Meat after 24 Hours Cooling (°F.).	Initial Temp. of Freezing Chamber (°F.).	Temp. of Freezing Chamber after 5 Hours (°F.).	(1) Temp. of Meat (°F.). (2) Temp. of Freezor (°F.). After 24 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 48 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 3 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 4 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 5 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 6 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 7 Days.	Part of Carcass. Weight (lb.).	Method used— I.—Iwanizky. K.—Keller.	Number of Cysts Swallowed.	Number of Cysts Evaginated.	Number of Cysts Digested (Dead).	Remarks Index.
11	5	43	19	14	(1) 28 (2) 10	(1) 22 (2) 12	(1) 23 (2) 19	(1) 14 (2) 14	(1) 12 (2) 11	— —	— —	Side of beef (288 lb.)	K.	13	1	12	5
12	4	42	21	—	—	(1) 23 (2) 16	(1) 19 (2) 11	(1) 20 (2) 15	— —	— —	— —	Hind quarter (160 lb.)	K.	10	4	6	4
13	6	41	23	15	(1) 21 (2) 18	(1) 19 (2) 17	(1) 20 (2) 16	(1) 19 (2) 17	(1) 18 (2) 15	(1) 17 (2) 11	— —	Side of beef (289 lb.)	K.	12	0	12	6
14	7	40	23	15	(1) 21 (2) 18	(1) 20 (2) 17	(1) 20 (2) 16	(1) 19 (2) 17	(1) 16 (2) 15	(1) 13 (2) 11	(1) 13 (2) 13	Side of beef (290 lb,)	K.	9	0	9	7
15	5	41	20	18	(1) 22 (2) 17	(1) 21 (2) 16	(1) 16 (2) 14	(1) 16 (2) 15	(1) 16 (2) 12	— —	— —	Side of beef (250 lb.)	K.	11	0	11	5
16	6	40	19	18	(1) 22 (2) 17	(1) 20 (2) 15	(1) 20 (2) 13	(1) 19 (2) 13	(1) 17 (2) 12	(1) 16 (2) 13	—	Side of beef (316 lb.)	K.	15	0	15	6
17	7	40	19	18	(1) 22 (2) 17	(1) 20 (2) 15	(1) 20 (2) 13	(1) 19 (2) 13	(1) 17 (2) 12	(1) 16 (2) 13	(1) 16 (2) 15	Side of beef (312 lb.)	K.	12	0	12	7
18	4	42	13	15	(1) 21 (2) 14	(1) 22 (2) 16	(1) 20 (2) 14	(1) 18 (2) 12	—	—	—	Side of beef (281 lb.)	K.	7	2	5	4
19	5	42	13	15	(1) 21 (2) 14	(1) 22 (2) 16	(1) 20 (2) 14	(1) 18 (2) 12	(1) 17 (2) 14	—	—	Side of beef (289 lb.)	K.	11	0	11	5
20	5	41	16	16	(1) 25 (2) 15	(1) 24 (2) 13	(1) 23 (2) 13	(1) 20 (2) 11	(1) 18 (2) 12	—	—	Carcass (614 lb.)	K. I.	34	0	34	5

TABLE C—(*continued*).

Experiment No.	Number of days in Freezer.	Temp. of Meat after 24 Hours Cooling (°F.).	Initial Temp. of Freezing Chamber (°F.).	Temp. of Freezing Chamber after 5 Hours (°F.).	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 24 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 48 Hours.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 3 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 4 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 5 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 6 Days.	(1) Temp. of Meat (°F.). (2) Temp. of Freezer (°F.). After 7 Days.	Part of Carcass. Weight (lb.)	Method used— I.—Iwanizky. K.—Keller.	Number of Cysts Swallowed.	Number of Cysts Evaginated.	Number of Cysts Digested (Dead).	Remarks Index.
21	1	—	16	16	(1) — (2) 15	—	—	—	—	—	—	Ox head of above	K.	10	0	10	1
22	6	42	17	18	(1) 23 (2) 14	(1) 22 (2) 15	(1) 21 (2) 13	(1) 20 (2) 13	(1) 21 (2) 14	(1) 19 (2) 13	— —	Carcass (572 lb.)	K. I.	30	0	30	6
23	6	41	15	16	(1) 25 (2) 16	(1) 23 (2) 14	(1) 23 (2) 12	(1) 19 (2) 13	(1) 18 (2) 15	(1) 18 (2) 13	—	Carcass (713 lb.)	K. I.	30	0	30	6
24	6	39	12	14	(1) 21 (2) 14	(1) 20 (2) 15	(1) 19 (2) 13	(1) 18 (2) 12	(1) 18 (2) 12	(1) 18 (2) 13	— —	Carcass (548 lb.)	K. I.	25	0	25	6
25	6	44	13	13	(1) 22 (2) 13	(1) 21 (2) 14	(1) 19 (2) 13	(1) 19 (2) 12	(1) 19 (2) 11	(1) 18 (2) 12	— —	Carcass (631 lb.)	K. I.	35	0	35	6
26	6	43	19	19	(1) 24 (2) 12	(1) 22 (2) 13	(1) 20 (2) 14	(1) 18 (2) 15	(1) 18 (2) 12	(1) 17 (2) 13	—	Carcass (651 lb.)	K. I.	30	0	30	6
27	6	42	18	17	(1) 21 (2) 9	(1) 20 (2) 13	(1) 18 (2) 13	(1) 17 (2) 13	(1) 18 (2) 17	(1) 18 (2) 14	—	Carcass (5[illegible]8 lb.)	K. I.	32	0	32	6
28	6	41	18	16	(1) 20 (2) 13	(1) 21 (2) 12	(1) 20 (2) 13	(1) 19 (2) 13	(1) 19 (2) 15	(1) 19 (2) 14	—	Carcass (686 lb.)	K. I.	30	0	30	6
29	6	42	13	13	(1) 21 (2) 14	(1) 20 (2) 14	(1) 20 (2) 13	(1) 20 (2) 11	(1) 21 (2) 13	(1) 19 (2) 9	—	Carcass (949 lb.)	K. I.	40	0	40	6
30	6	38	11	12	(1) 21 (2) 16	(1) 20 (2) 15	(1) 20 (2) 16	(1) 20 (2) 15	(1) 19 (2) 14	(1) 19 (2) 15	—	Carcass (548 lb.)	K. I.	35	0	35	6
31	6	44	16	16	(1) 22 (2) 14	(1) 21 (2) 13	(1) 20 (2) 14	(1) 21 (2) 16	(1) 20 (2) 14	(1) 19 (2) 11	—	Carcass (438 lb.)	K. I.	30	0	30	6

did not consider that any useful purpose was served by using our available material on chilling tests. Secondly, it was very undesirable to keep rapidly decomposing beef in the condemned meat section of the abattoir chilling rooms.

Our viability tests with beef measles were, therefore, mainly confined to freezing tests, the results of which are tabulated in Table C.

As with *C. cellulosae*, we performed a number of contemporary tests in 5 per cent. sodium taurocholate solution, and in the case of *C. bovis*, with 30 per cent. ox bile-physiological saline solution, instead of pig bile. Viability of the fresh measles was always tested by Keller's method, prior to the carcass being placed in the freezing chamber. Twenty-four hours' cooling of the carcass was allowed in every case, prior to freezing.

Remarks Index to the Foregoing Table (C).

1. *Experiment No.* 1.

A side of beef weighing 248 lb. was frozen for 24 hours. In that time it was found that the beef was by no means frozen through, although freezing had penetrated a considerable distance into the deeper tissues. Twenty measles were removed from the deeper shoulder and thigh muscles. Our subject, W.H.G., swallowed four in two tubes according to our modification of Keller's method. All four scolices evaginated.

Contemporary tests were done as follows, in an incubator with governed temperature of 38° C:—

In 5 per cent. sodium taurocholate solution, 9 out of 12 scolices evaginated.

In 30 per cent. ox bile-saline solution, 1 out of 4 scolices evaginated.

Experiment No. 4.

A relatively heavy (361 lb.) side of beef was frozen for 24 hours. Here again, freezing was not quite complete. Our subject swallowed 10 measles in 4 tubes. All scolices evaginated.

Experiment No. 7.

A side of beef weighing 245 lb. was frozen for 24 hours. The same remarks apply, as above. Subject swallowed 8 *cysticerci* in 4 tubes. All scolices evaginated.

Experiment No. 21.

An ox head containing some 20 viable measles was frozen for 24 hours. Ten of these measles were selected, since they were "covered" up by the flaps of the masseteric incisions Our subject swallowed these 10 and all were found to be dead.

Summary.

Twenty four hours' freezing of beef carcasses split into halves (sides of beef) is not sufficient to kill *C. bovis*, since, in that time the freezing process has not had sufficient time to permeate into the deeper musculature. Shallowly situated measles such as those in the masseters are killed with ease after 24 hours' freezing.

For illustrations see Figures 8 to 11.

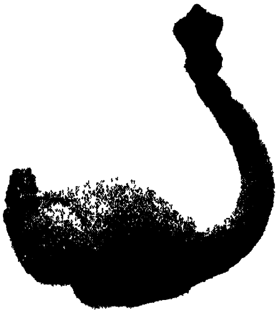

FIGURE 8.

Fresh *C. bovis* scolex evaginated in two hours in 5 per cent. Sodium taurocholate solution. Magn. 7×.

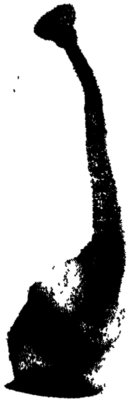

FIGURE 9.

Fresh *C. bovis* scolex evaginated in 2 hours in 30 per cent. ox bile-physiological saline solution. Magn. 7×.

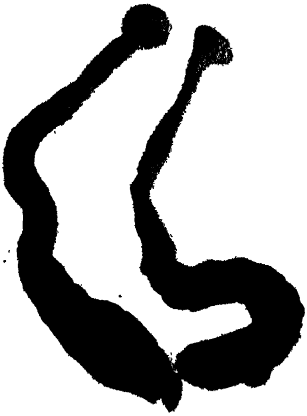

FIGURE 10.

C. bovis scolices evaginated by Keller's method after 24 hours freezing. Magn. 7×.

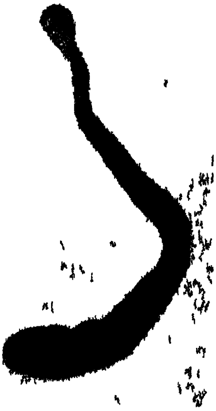

Figure 11a.

C. bovis scolex evaginated by Keller's method after 24 hours, freezing. Magn. 7×.

Figure 11b

C. bovis head, from scolex illustrated in Figure 11a. Note four suckers plainly visible. Magn 40×.

2. *Experiment No. 2.*

Side of beef weighing 262 lb. was frozen for 48 hours. At the end of that time the temperature of the deeper tissues of the buttock had reached 27° F. (approximately -3° C.), and the room temperatures had fallen from 17° F. to 13° F. (approximately -10.5° C.). Freezing had, by that time, permeated completely into the deeper tissues.

A remarkable phenomenon occurred in this test, in so far as that neither of two *cysticerci* swallowed by our subject evaginated their scolices, whereas in 5 per cent. sodium taurocholate solution 4 out of 10 scolices evaginated in 2 hours, and in 30 per cent. ox bile-physiological saline solution 3 out of 10 scolices evaginated in 2 to 4 hours.

Experiment No. 6.

A side of beef weighing 254 lb. was frozen for 48 hours. Same remarks regarding freezing apply in this case as above. One out of 12 scolices evaginated by Keller's method.

Experiment No. 3.

An ox tongue was frozen for 48 hours. Freezing was definitely complete. No scolices from 8 measles evaginated by Keller's method.

Summary.

After 48 hours' freezing in sides of beef weighing approximately 260 lb., the freezing process has permeated considerably into the deeper musculature. The freezing action has, however, not yet had sufficient time to destroy the viability of *C. bovis*. In small pieces of meat, for example an ox tongue, the freezing process is definitely complete, and few measles will survive the low temperature reached in the interior of such meat.

Illustrations see Figures 12 and 13.

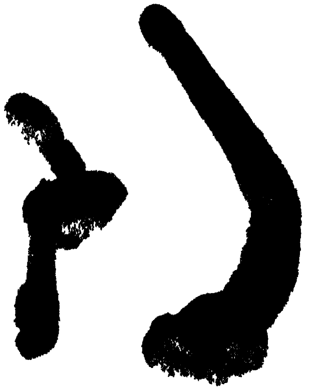

FIGURE 12.

C. bovis scolices evaginated in 2 hours in 5 per cent. sodium taurocholate solution. Measles from carcasses frozen for 48 hours. Magn. 7×.

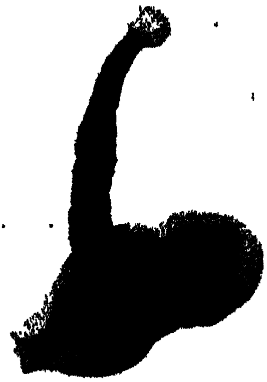

FIGURE 13.

C. bovis scolices evaginated in 2 hours in 30 per cent. ox bile-physiological saline solution. Measles from carcasses frozen for 48 hours. Magn. 7×.

3. *Experiment No. 5.*

A fairly heavy (350 lb.) side of beef from the same carcass as that of Experiment No. 4, was frozen for three days. After three days' freezing the inner temperature of the buttock, read on a " spear thermometer ", registered 20° F. (approximately - 7° C.). By that time freezing was complete. After 24 hours' thawing of the meat, W.H.G. swallowed 7 *C. bovis* in three tubes. All seven scolices evaginated. Contemporary artificial evagination tests were tried. Five *C. bovis* were treated in each of a 5 per cent. sodium taurocholate solution and a 30 per cent. ox bile-saline solution. Negative results were obtained.

Experiment No. 8.

A side of beef weighing 250 lb., from the same carcass as that described in Experiment No. 7, was frozen for 3 days. The inner temperature of the meat fell from 25° F. after 24 hours to 23° F. after three days. Freezing room temperature varied between 16° F. and 11° F. Freezing was complete. Out of 12 measles swallowed by W.H.G. in four tubes, no less than 9 evaginated their scolices beautifully. Contemporary artificial tests with 10 measles in each sodium taurocholate solution and 30 per cent. ox bile-saline solution, gave negative results.

Experiment No. 9.

A side of beef weighing 248 lb. was completely frozen through in three days. Inner temperature of the meat fell from 28° F. after 24 hours to 23° F. after 3 days. Room temperature fell from 18° F. after 24 hours to 11° F. after 3 days. Out of 10 measles swallowed by W.H.G., two evaginated their scolices.

Summary.

Three days' freezing of sides of beef is not sufficient to kill *C. bovis*. A temperature of 23° F. is easily maintained in the meat after 24 hours, provided the room temperature is kept below - 10° C.

4. *Experiment No.* 10.

A side of beef weighing 300 lb. was frozen through for 4 days. The internal temperature of the meat registered 28° F. after the first 24 hours' freezing, and fell rapidly between the third and fourth days from 23° F. to 14° F., which temperature was the same as that of the freezing chamber. On the third day the temperature of the freezing chamber necessarily rose to 19° F., as the result of the opening of the chamber for about an hour, in order to take out, and place in a few lightly infested measly carcasses for Regulation freezing treatment. After 24 hours' thawing of the meat, W.H.G. swallowed three tubes containing 10 measles. Of this number four scolices evaginated. Contemporary tests with sodium taurocholate solution and ox bile-saline solution gave negative results.

Experiment No. 12.

A hind-quarter of beef weighing 160 lb. was frozen for 4 days. The initial temperature of the freezing chamber was 21° F., but after the quarter had been in the chamber 48 hours, the temperature had fallen to 16° F., and this was succeeded on the third and fourth days by 11° F. and 15° F. respectively. The internal temperature of the meat after the 2nd, 3rd and 4th days was 23° F., 19° F., and 20° F., respectively. Freezing of the quarter was complete. After 24 hours' thawing of the meat, W.H.G. swallowed 10 cysts, of which number 4 evaginated scolices.

Experiment No. 18.

A side of beef weighing 281 lb. was frozen for 4 days. Freezing was complete, and on no occasion during that time did the freezing-room temperature exceed a maximum of 16° F. (approximately - 9° C.). The internal temperature of the meat fell from 23° F. (approximately - 6·5° C.) after the first 48 hours' freezing to 18° F. (approximately - 8° C.) after the 4th days' freezing. After 24 hours' thawing of the meat W.H.G. swallowed seven measles in 3 tubes. Of this number two evaginated their scolices. Contemporary tests with sodium taurocholate solution and bile-saline solution gave negative results.

Summary.

Out of a total of 27 measles from meat frozen for four days, swallowed by W.H.G., ten were still capable of evaginating their scolices under conditions tantamount to natural infection. Freezing in all cases was complete, and a uniformly low temperature was maintained. *Cysticercus bovis* is not destroyed by thorough freezing in all cases within four days. Illustrations see Figure 16.

5. *Experiment No.* 11.—A side of prime "young" beef weighing 288 lb. and containing a large quantity of fat, uniformly distributed, was frozen for 5 days. This side of beef was from the same carcass as that of Experiment No. 10. That freezing was complete can be gleaned from the following temperature records, which are specially repeated in this case:—

	Initial Temp. of Freezer.	Temp. after 5 Hours.	Temp. after 24 Hours.	Temp. after 48 Hours.	Temp. after 3 Days.	Temp. after 4 Days.	Temp. after 5 Days.
	°F.	°F.	°F.	°F.	°F.	°F.	°F.
(1) Of Meat....	—	—	28	22	23	14	12
(2) Of Chamber	19	14	10	12	19	14	11

Only on one occasion, as was noted under the description of Experiment No. 10, did the freezing-room temperature exceed −10° C. for the reasons stated. It took forty-eight hours before sufficient thawing had occurred to permit of the side of beef being dissected in order to collect deep-seated *cysticerci*. Thirteen measles were given to W.H.G. to swallow in 4 tubes. Of this number one evaginated its scolex. I sent this evaginated scolex to Dr. H. O. Mönnig, Onderstepoort, with a request that he should examine and describe its condition, and, if necessary prepare it for photographing purposes. Dr. Mönnig kindly favoured me with the following report:—

"*Scolex of C.* bovis *evaginated after* 5 *days' freezing.*

The scolex is provided with two apparently normal suckers situated on two adjoining quarters. The other two quarters are slightly thickened, as if rudimentary suckers were present, but no such structure is visible. Between the latter two quarters there is a large invagination of which the opening is as wide as a normal sucker. The lumen of the invagination is then constricted, but widens out again to form a fairly large space in the centre of the scolex, from which two arms are given off in the directions of the two normal suckers and almost reaching these. The orifice of the invagination is lined with cuticle bearing a number of evenly-spaced striations directed from without inwards. The walls of the invagination within the scolex are thrown into a number of large folds. It is problematic whether the scolex could have attached itself normally to the intestinal wall and, if it could have done so, it is probable that the tapeworm would have had some difficulty in maintaining its position after it had grown to some length." Illustration of this scolex, see Figure 17.

Experiment No. 15.

Side of beef weighing 250 lb. was frozen for 5 days. Freezing was again complete and uniformly low temperatures were maintained. W.H.G. swallowed 11 measles, of which number none evaginated scolices.

Experiment No. 19.

Side of beef weighing 289 lb. was frozen for 5 days. This side was from the same carcass as that of Experiment No. 18. Out of 11 measles swallowed, no scolices evaginated.

Experiment No. 20.

Carcass of beef weighing 614 lb. was completely frozen through for 5 days. Note the low temperatures maintained of (1) the meat and (2) the freezing chamber. After 48 hours' thawing of the meat W.H.G. and two others swallowed 34 measles. Of this number no scolices evaginated. (One bag containing 3 measles was " lost ".)

Summary.

Out of a total of 69 measles swallowed by my assistants, one evaginated its scolex, after five days' freezing. Dr. Mönnig has aptly described the deformities of the head of this evaginated scolex, but, nevertheless, if we must accept the theory of complete evagination of the scolex as the best criterion of viability of measles, then we must bear in mind that occasionally viable *cysticerci* may survive in beef frozen for five days.

6. *Experiments Nos.* 13 *and* 16.

Sides of beef weighing 289 lb. and 316 lb. respectively, were completely frozen through for six days. Out of a total of 27 measles swallowed by my assistant, W.H.G., no scolices evaginated.

Experiments Nos. 22 *to* 31.

Having established the possibility that *C. bovis* might occasionally survive five days' continuous freezing, I decided to confine my tests after the twentieth experiment to measles from carcasses frozen for six days. Consequently, the last ten carcasses we obtained during the months of December, 1936, and January and February, 1937, were subjected to six days' continuous freezing. As many *cysticerci* as possible were collected from both sides of those respective carcasses, after the latter had been sufficiently thawed. The *cysticerci* were enclosed in tubes and silk bags and were swallowed by W.H.G. and two assistants. W.H.G., as usual recovered all his tubes, but a few of the silk bags were " lost " by his two confréres. Nevertheless, out of a total of 317 measles collected from 10 carcasses, no scolices evaginated.

7. *Experiments Nos.* 14 *and* 17.

Two fairly large sides of beef, weighing 290 lb. and 312 lb., respectively, were completely frozen for 7 days. Out of a total of 21 measles swallowed by W.H.G., no scolices evaginated.

The following short table summarizes the results of our freezing tests with *C. bovis*. (Sides, Quarters and Carcasses of Beef only): —

Days Frozen.	Total Measles Swallowed.	Total Scolices Evaginated.	Digested or "Lost."	Percentage Viable.
1	22	22	0	100·0
2	14	1	13	7·1
3	29	18	11	62·07
4	27	10	17	37·04
5	69	1	68	1·45
6	344	0	344	—
7	21	0	21	—

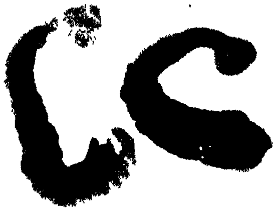

FIGURE 14.

C. bovis scolices evaginated by Keller's method after 3 days' freezing. Magn. 7×.

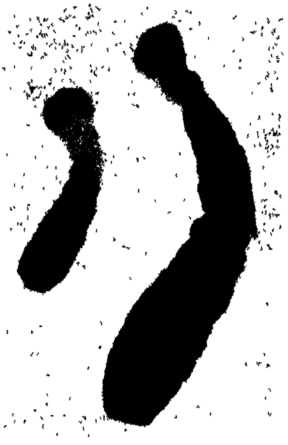

FIGURE 15.

C. bovis scolices evaginated by Keller's method after 3 days freezing. Magn. 7×.

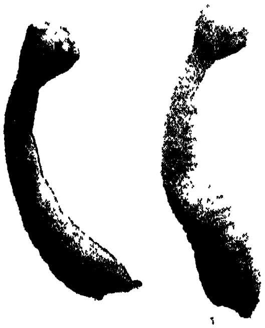

FIGURE 16.

C. bovis scolices, evaginated by Keller's method after 4 days' freezing. Magn. 7×.

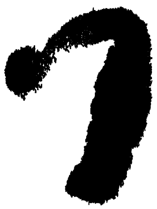

FIGURE 17A.

C. bovis scolex evaginated by Keller's method, after 5 days' continuous freezing. Magn. 7×.

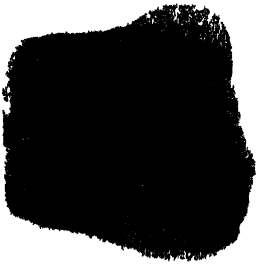

FIGURE 17B.

Microscopic view of same scolex. See description of Experiment No. 11 Magn. 40×.

CONCLUSIONS.

From the results we obtained with the available material at Bloemfontein Abattoir, it is reasonable to conclude that *C. bovis,* frozen in whole sides of beef can withstand a considerable amount of freezing. That the beef *cysticerci* can remain viable under those circumstances, after 24 hours' to 4 days' continuous freezing, is without much doubt, if the fullest recognition of our criterion of simulating natural infection is to be accepted as the most conclusive evidence thus far provided by modern science.

Our table, giving the most meticulous daily temperature recordings, shows that the most thorough through freezing of the tested carcass was applied.

After five days' continuous freezing, on one occasion, my subject recovered an evaginated scolex. This scolex, as was described by Dr. Monnig, was to a great extent deformed in the head process, and only two normal suckers could be noticed. Since malformations are not altogether unusual in *cysticerci*, it may be considered possible that this particular *cysticercus* may have evaginated a deformed scolex under normal and fresh conditions. It may also not be unreasonable to presume, that since one deformed *cysticercus* out of a not excessive number tested (69), was capable of evaginating its scolex, a few quite normal *cysticerci* might be able to do so, if swallowed by humans with the ingestion of measly beef frozen for five days. We may, therefore, reasonably presume that a five days' freezing treatment is risky, if we are to satisfy ourselves of the shortest period of freezing necessary for the destruction of *Cysticerci bovis.*

Superficial *Cysticerci bovis*, e.g. those situated in the tongue, or in the masseters of the face, are destroyed within 48 hours, according to our tests. Provided a temperature of -10° C. is maintained for that period, through freezing of a relatively thin muscular organ, like the tongue, or of a thin flap-like muscle, like the masseter, will occur with certainty.

Judging from our tests, it would appear that no *Cysticerci bovis*, situated in the deep musculature of the shoulders or of the thighs can survive a period in excess of five days' freezing. After 6 days' freezing, 344 deep-seated measles (shallow measles were ignored and not used) failed to evaginate their scolices, when swallowed according to our modifications of Keller's and Iwanizky's methods. Similarly, we had negative results with 21 measles tested on two occasions after 7 days' freezing. If our results, offered not in a dogmatic manner, were to be accepted, it can reasonably be presumed that no *cysticerci* will remain viable after six days' continuous freezing, but there is a definite probability that a few individual deep-seated *cysticerci* can survive a five days' freezing.

The South African Regulations provide for a period of 14 days' continuous freezing at - 10° C., and there is no doubt at all, that this period is perfectly safe from the public health point of view. If any good purpose were to be served thereby, e.g. economy to the butchers, I feel that the period of freezing can with a margin of safety, be reduced to 10 days' freezing. The only objection I have to a ten days' freezing process is that, unless the abattoir is a large concern and several freezing chambers may be available, it will be difficult to organise control and maintenance of low temperatures. This can be elucidated by the following example. At Bloemfontein we only have one freezing chamber available at the present time. In order to maintain the low temperature required, we have set aside one morning a week (every Thursday), when we take out of, or place into, the freezing chamber the week's number of lightly infested carcasses, which, meanwhile, have been collected and kept in a part of the chill-rooms set aside for that purpose. This operation takes about an hour every week, and after that the freezing chamber is kept locked until the next week, except for a few minutes on certain occasions during the past year, when we were conducting our viability experiments. By setting aside a certain day once a week for working in the freezing chamber, a regular control can be exercised, whereas with ten days' freezing, irregular days of taking out or putting in carcasses will follow.

In agreement with Keller, one must stress the fact that low temperatures must be maintained, otherwise, as Keller found, all *cysticerci* may not be killed even after about 23 days' freezing at such high temperatures of, e.g. - 1° C. to - 1·5° C. (±30° F.). (See Table on next page.)

PART V.

The Importance of Cysticercosis in Meat in Relationship to Public Health.

It has been considered a moot-point as to whether the *Cysticercus cellulosae* in pork and its correlative *Taenia solium* in man, or the *Cysticercus bovis* and its adult *Taenia saginata* is the more important parasite, from the perspective of public health. At the present time it would appear that *Taenia saginata* is far more frequently found in humans than *Taenia solium*, in most countries where both parasites are encountered. Yet, in many countries, and in South Africa in particular, *Cysticercus cellulosae* in the pig is the more common larval form.

COMPARATIVE TABLE SHOWING THE RESULTS AND RECOMMENDATIONS OF VARIOUS WORKERS.

Temperatures and Periods of Freezing Lethal to Cysticerci.

Investigator.	*Cysticercus cellulosae.*			*Cysticercus bovis.*		
	Temperature Required.	Time.	Part of Carcass.	Temperature Required.	Time.	Part of Carcass.
Reissmann (1897)...........................	—8°C. to —10°C.	4 days	Depths	—8°C. to —10°C.	3 days	Depths.
Boccalari (1903)............................	—4°C. to — 6°C.	4 days	—	—4°C. to —10 °C.	4 days	—
Ransom (1914)...............................	—	—	—	—9°C. to —12°C.	6 days	Quarters.
Killisch (1923)...............................	—8°C. to —12°C.	3½ days	Half-pigs	—	—	—
Wagner (1922)...............................	—	—	—	—	8 days	Carcasses.
Schmey & Bugge (1930)......................	—8°C. to —10°C.	4 days	Carcasses	—8°C. to —10°C.	4 days	Carcasses.
Kallert (1931)...............................	—	—	—	—8°C. to —10°C.	9–10 days	Hindquarters.
Clarenburg (1932)............................	—	—	—	—8°C. to —10°C.	65 hours	6 cm. pieces.
Feldförth (1934).............................	—	—	—	—2°C.	2 days	½-lb. weight.
*Scheerer (1935).............................	—	—	—	—2°C.	6 days	Innermost parts.
†Zunker (1935)...............................	—	—	—	—3°C.	6–7 days	Innermost parts.
Bloemfontein Abattoir (1936–37)..............	—10°C.	5 days	Carcasses	—10°C.	6 days	Carcasses.

* Scheerer explained that a temperature of —2°C. must be maintained for 6 days in the innermost parts of the carcass.

† Zunker maintained that a temperature of —3°C. in the innermost parts of carcass was lethal to *C. bovis* and it generally took 6 to 7 days to reach —3°C. in those parts.

It has hardly been found practically possible to conduct scientific surveys of the incidence of the two species causing human taeniasis in any country, and, therefore, recent literature is remarkably silent on the question. That either or both parasites occur almost universally may be gleaned from reference to the recent articles by Liévre (France, 1933); Cameron (Great Britain, 1933), Robertson (Great Britain, 1920); Krueger (1934), Junack (1926-31), Profé (1934) and many others in Germany; van der Slooten (1932), van Oijen (1929), Kerstens (1931), and many others in Holland; Cattaneo (1932) and others in Italy; Krupski (1917), Guillebeau (1917) and others in Switzerland; Dikoff (Bulgaria, 1931); Elvinge (1929) and Nielsen (1934) in Denmark; Grado (Sicily, 1935); Michail (Roumania, 1935); Eguchi and Nishiyama (Japan, 1930); Mills (1923-24), Gear and Pedersen (1934) in China; Rao (India, 1933); Yenikomshian and Berberian (Syria and Lebanon, 1934); Bergeon (French Indo-China, 1928); Le Coultre (Dutch East Indies, 1928); Price (U.S.A., 1925), Hall (U.S.A. and Central America, 1927); Nauck (Costa Rica, 1931); Palais (Brazil, 1933); Schwartz (1925) and Schwartz and Tubangui (1922) in Phillipine Islands; Penfold, Penfold and Phillips (1936) in Australia; Claverie (French Guinea, 1928); Teppaz (Senegal, 1923); Maplestone (Sierra Leone, 1924); Poisson (Madagascar, 1929 and 1934); Daubney and Carman (Kenya, 1928); Porter (1918), Watkins-Pitchford (1923), Cawston (1934 and 1935) and Mönnig (1934 and 1936) in South Africa.

A detailed survey of the incidence of human taeniasis is not given in every case by the above authors, in respect of the countries to which the various articles refer, but the extent of infection in some countries can be gauged from extracts from many of the articles According to Liévre (1933), in France in every hundred cases of tapeworm infection, only one is due to *Taenia solium*. The frequency of human infection is inversely proportional to the degree of infestation of the intermediate host.

According to Junack (1926) infestation of humans with *Taenia saginata* in Germany had a big increase during the war period, when many soldiers served in parts where no meat inspection existed, or where the inspection may have been of a perfunctory nature.

Special reference may be made to countries with predominantly non-European populations. Eguchi and Nishiyama (1930) report that *Taenia solium* is very rarely met with in Japan, with the exception of the Prefecture of Okinawa, where they found twenty-five cases of *T. solium*. Wu (1936) stated that in all, fifteen cases of *Taenia solium* have been reported from the following Chinese provinces: Shantung 1, Hupeh 1, Hunan 1, Yunnan 1, Kwangtung 1, Shansi 1, Szechuan 3, Hopei 5, Chekiang 1.

According to Wu, fifteen cases of *Taenia saginata* were also reported from the following Chinese provinces: Hopei 12, Yunnan 1, Anhwei 1, Fukien 1.

Mills (1924) stated that in 2 years he treated 12 patients for *Taenia saginata* in the Peking Union Medical College, including one American girl of 24. No cases of *Tacnia solium* were observed by Mills. He believed that taeniasis was much more prevalent in

China than was supposed. Although very few patients were actually treated in clinics, a vast number was treated by native medicines in bazaars.

Liang (1932), quoted by Gear and Pedersen (1934), reported a case of *Taenia solium* infection, and in the Chinese hospital survey only two cases of *T. solium* were specifically diagnosed, one from Peiping and one from Nanking, both in Chinese subjects.

Rao (1933) reported a case of *T. solium* in Madras, India, and made special mention that he believed the *T. solium* was a more common parasite in India than has been revealed.

That *Taenia saginata* is quite a common parasite in Syria and Lebanon, is shown in the statistics supplied by Yenikomshian and Berberian (1934). These authors state that " taeniasis is much more common in Beirût and its surroundings than in Aleppo and Damascus. In both Syria and Lebanon, meat is frequently eaten raw as *Kibbi neyyi*, a national dish, or broiled. In Beirût more beef is eaten than in Aleppo and other parts of Syria, where mutton and goat meat is preferred ". In the four main cities of Syria and Lebanon and on the Amik plain the authors found the incidence of *T. saginata* in faecal examination to be: Aleppo 2·6 per cent., Damascus 3 per cent., Baalbek 3 per cent., Hamah 0 per cent. and Amik plains 5 per cent. In Beirût the incidence was found to be 12 per cent. In that particular area no *T. solium* was found, since the majority of the inhabitants were Mohammedans, who did not eat pork. On the coast belt and along the Orontes River the incidence of *Taenia saginata* was 10 per cent.

Penfold, Penfold and Phillips (1936) give a suggestion of the extraordinary incidence of *T. saginata* infection among Syrians. These authors conducted a survey of the incidence of tapeworm infection in the State of Victoria, Australia, and they found that 90 out of 1,830,000 people in that State had *Taenia saginata*. Of that number 42 were Syrian-born Australians. In the entire State of Victoria there were only 377 people who were born in Syria, and 42 were infected, or 11,000 per 100,000. The survey was conducted under the aegis of the Victoria Government, who offered a reward of £5 to carriers for the production of a complete *Taenia*. In addition a very thorough questionnaire was sent to all physicians and chemists.

According to Bergeon (1928) and le Coultre (1928), both parasites are relatively common among natives in French Indo-China and in Bali, respectively.

Mr. J. T. Forbes, M.R.C.V.S. of Singapore writes (19.11.36) that medical authorities have reported a very low incidence of taeniasis in Malaya, but Mr. Forbes has " reason to believe that the incidence of infection is considerably higher than is anticipated ".

In the Phillipine Islands, according to Schwartz and Tubangui (1922), the incidence of *T. saginata* was relatively high. About 30 in 4,000 stool examinations were positive. The incidence of *T. solium* was much lower.

Price (1925) believed that the incidence of *T. solium* was high in Texas, U.S.A., with its large Negro and Mexican population.

Nauck (1931) found that *Cysticercus cellulosae* in humans, due to the frequency of *T. solium* among the inhabitants was very readily acquired in Costa Rica.

In order to obtain knowledge of the incidence of infection with various kinds of worms among natives in Sierra Leone Protectorate, Maplestone (1924) examined the stools of 500 natives, inmates of the Freetown gaol. He found that 3·2 per cent. were infected with *T. saginata.*

Daubney and Carman (1928) examined the stools of the inmates of a Government reformatory in the Kenya Highlands and found the incidence of *T. saginata* to be 50 per cent. The inmates of this reformatory were boys drawn from all parts of East Africa, and represented almost all tribes.

Capt. H. J. Lowe, M.R.C.V.S., of the Department of Veterinary Science and Animal Husbandry, Mpwapwa, Tanganyika Territory, supplies copies of reports from some Medical Officers concerning the incidence of human tapeworm infestation in different parts of the Territory. (Letter dated 24.10.36):—

Dr. R. C. Speirs, Medical Officer, Moshi found 313 infected stools among 552 examinations from prisoners, sanitary porters, school boys and other native children. (April 1933.)

Dr, W. Hood-Dye, Medical Officer, Iringa found 34·17 per cent. of stool examinations positive for *T. saginata* among the Wahehe tribe, and 14·02 per cent. among the Wabena tribe. (August 1933.)

Dr. A. McA. Blackwood, Medical Officer, Dodoma, found among inpatients at his hospital that 34 out of 638 stool examinations were positive for *T. saginata*, or 5·32 per cent. (July 1933.)

Dr. J. S. Armstrong, Medical Officer, Singida, treated 2,456 cases for tapeworm at the Singida Hospital during the five years 1928-32. He calculated that the Singida Hospital served a population of 45,000-50,000 people. (May 1933.)

Dr. D. A. Skan, Medical Officer, Dar-es-Salaam, found 110 stool examinations out of 3,015 positive for *Taenia* infection. (April 1933).

We have already seen that many of the earlier writers mentioned the severe—almost 100 per cent.—incidence of *T. saginata* among the Abyssinians. (Reference Neumann, 1892; Leuckart 1886). To what extent the infection occurs in that country at the present time is not known, since little or no literature is available on the subject.

Poisson (1930) supplied statistics for the year 1927 of recorded cases of *T. solium* in Madagascar. He mentioned that the bulk of cases found in Madagascar were residents of the *hovas*, also among Europeans born in Reunion, and the disease was not unknown among Europeans resident in the Capital, and common among Indians at Farafangana.

According to Poisson, 49 cases of *T. solium* were recorded in Madagascar in 1927, of which number, 16 came from Farafangana, and 6 among the *tirailleurs* of the garrison at Majunga.

It is a great pity that regular surveys of the incidence of taeniasis are not undertaken in our civilized countries. In South Africa, Porter (1918) did a survey of the incidence of helminthic infection among natives in Johannesburg. In the Johannesburg

General Hospital she detected the ova of tapeworms in the excretum of 37 out of 375 native patients, and in 1 out of 60 European patients. "All of these patients had been admitted for diseases other than 'worms', and many of them were surgical cases." Twenty-six of the natives harboured *T. saginata* and eleven *T. solium*. The European case harboured *T. saginata*. Porter further recorded 104 post-mortem examinations on native mine labourers. "Tapeworms were discovered in the intestinal canal in 20 instances. (12 *T. saginata* and 8 *T. solium*.)"

Watkins-Pitchford (1923) estimated the incidence of tapeworm infection among South African natives to be from 10 to 19 per cent., "and it is not uncommon amongst Europeans". According to Watkins-Pitchford, between 1917 and 1923, 17 cases of tapeworm (12 *T. saginata* and 5 *T. solium*) were diagnosed in Europeans from microscopic examination of faecal specimens sent to the South African Institute for Medical Research, Johannesburg. "Such returns do not give, of course, any indication of the extent of the prevalence among Europeans. Many people harbour these parasites and are quite unconcious of the fact, because they never inspect their own dejecta. Those cases in which diagnosis is arrived at by microscopic examination of faecal specimens must represent a very small fraction of the total number."

Dr. A. J. Orenstein, Chief Medical Officer, Rand Mines, Ltd., very kindly supplied the following tables which show the incidence of worm infestations among natives employed on the City Deep Mine, Johannesburg. The tables were compiled by Dr. W. O. Fischer, for the years 1928-33, inclusive.

TABLE I.

Incidence of Tapeworm in 1,086 consecutive autopsies on Native Mine Workers of the City Deep Central Native Hospital.

Tribe.	No. of P.M.'s.	*Taenia saginata.*		*Taenia solium.*	
		No.	Percentage.	No.	Percentage.
Shangaan	333	4	1·2	—	—
Mchopi	170	5	2·9	—	—
Nyambaan	116	3	2·6	—	—
Tonga	32	2	6·3	—	—
EAST COAST NATIVES TOTAL	651	14	2·3	—	—
Basuto	215	5	2·3	1	0·47
Xosa	93	1	1·1	—	—
Zulu	34	2	5·9	—	—
Bechuana	29	—	—	—	—
Pondo	27	1	3·7	—	—
Fingo	9	—	—	—	—
Hlubi	9	2	22·2	1	11·1
Baca	8	2	25·0	—	—
Moenda	5	—	—	—	—
Swazi	4	3	75·0	—	—
Cape Coloured	2	—	—	—	—
UNION NATIVES TOTAL	435	16	3·7	2	0·5
TOTAL	1,086	30	2·8	2	0·2

TABLE II.

Showing incidence of Tapeworm ova in the stools of Natives of the City Deep Mine in 1,016 *consecutive examinations.*

Tribe.	No. of Stools Examined.	Ova of *Taenia saginata.*		Ova of *Taenia solium.*	
		No.	Per-centage.	No.	Per-centage.
Shangaan	214	7	3·3	1	0·47
Nyambaan	38	1	2·6	—	—
Mchopi	31	2	6·5	—	—
Tonga	20	—	—	—	—
EAST COAST NATIVES TOTAL	303	10	3·3	1	0·47
Basuto	352	16	4·5	2	0·6
Xosa	145	4	2·8	1	0·69
Bechuana	68	1	1·5	—	—
Pondo	44	1	2·3	—	—
Zulu	36	—	—	—	—
Swazi	34	1	2·9	—	—
Baca	9	1	11·1	—	—
Fingo	8	—	—	—	—
Moenda	7	—	—	—	—
Hlubi	5	—	—	—	—
Cape Coloured	4	—	—	—	—
UNION NATIVES TOTAL	713	24	3·4	3	0·42
TOTAL	1,016	34	3·3	4	0·4

Since the above, according to Dr. Orenstein, Dr. Fischer in 1935 found *Taenia saginata* in 2·2 per cent. of 934 Union Natives (excluding Zulus); in 3·9 per cent. of 103 Zulus; and in 3·3 per cent. in East Coast Natives.

Note: By *East Coast Natives* is meant those from Portuguese East Africa.

Dr. C. G. Becker (17.2.37) kindly supplied statistics showing the number of positive examinations of the stools at the South African Institute for Medical Research, Johannesburg, for the years 1934 to 1936 (inclusive). The following table illustrates the numbers of cases found (ova and/or segments), and the statistics refer to *T. saginata*, except where special mention is made of *T. solium*.

Year.	Europeans.		Non-Europeans.		Total Stools Examined.
	Ova.	Segments.	Ova.	Segments.	
1934	14	25	21	9	4,700
1935	9	19 plus 1 *T. sol.* (segments)	11	3	3,844
1936	11 plus 1 *T. sol.* (ova and segs.)	13	30	2	3,813

Between the period 4.4.36 and 10.1.37, the following cases of Taeniasis were treated at the Pretoria Hospital. (Statistics kindly supplied by Dr. H. J. Hugo, Medical Superintendent.)

Europeans.

Age.	Sex.	Diagnosis.	Date.	Remarks.
11	F.	*T. solium*	4.4.36	
6	M.	„	20.8.36	
10	F.	„	7.9.36	One family, N.B.
3	M.	„	7.9.36	
8	F.	„	7.9.36	
4	F.	„	30.9.36	
5	M.	„	23.10.36	
Non-Europeans.				
5	F.	*T. solium*	5.1.37	Basuto. Admitted in coma.
14	F.	„	10.1.37	Basuto. Admitted as accident case.

Judging from these statistics, and from figures supplied by the medical authorities of two of our neighbouring Native Protectorates, it would appear that the incidence of taeniasis among natives and Europeans is not high in Southern Africa. Dr. J. W. Stirling, Principal Medical Officer, Bechuanaland Protectorate reports (4.11.36): "From reported cases one would conclude that infection in humans of tapeworms is not excessive. Out of 27,662 first attendances of out-patients in 1935, only 43 were for tapeworm infection." Dr. H. W. Dyke, Principal Medical Officer, Basutoland, reports (30.10.36): "Out of 133,021 out-patients there were 231 cases of tapeworm. These figures are for a three-year period at all Government stations. At Maseru out-patient department for the period 1st January to 30th September, 1936, out of 7,800 out-patients, there were 7 cases of tapeworm ".

There can be little doubt that the figures given in the statistics, and also those of the two Native Protectorates are only indicative of a very small percentage of the actual infections. Reference to the Survey of the Incidence of Cysticercosis in an earlier Part of this work shows a particularly high incidence of *C. bovis* in Natal, where a large number of cattle of Zulu origin is slaughtered; in Barberton, which district borders on Swaziland; and in the coastal Cape-Eastern abattoirs, East London, Kingwilliamstown, Fort Beaufort and Port Elizabeth, centres which draw a good deal of their stock from the Transkeian Native Territories. The Eastern Orange Free State is a " black " area as regards *C. cellulosae.* Reference to the " Incidence Map " shows the proximity of the districts of Ficksburg, Senekal, Clocolan and Wepener to the Basutoland border. We have noted that the Principal Veterinary Officer for Basutoland estimated that the incidence of *C. cellulosae* in that Territory was about 10 per cent. in pigs. South African Statistics—the few which are available and not by any means truly indicative—appear to agree with the observations of Lièvre in France, that *T. saginata* is encountered far more frequently than *T. solium*, and that the incidence of infection in the human is inversely proportional to the severity of the infection in the intermediate stage. Undoubtedly, if pork were not eaten in a well-cooked state, even by natives, the incidence of *T. solium* would be much higher everywhere in South Africa, but the omnivorous habits of the pig tend to the ready ingestion of the entire human stool, containing thousands of *T. solium* ova, and heavy infestation of the pig follows. A theory, which I think bears a good deal of fact and may possibly explain the somewhat anomalous disproportion between the relative frequency of infection in the pig and the rarer infection of the human in South Africa, with the adult tapeworm, *Taenia solium*, is that the risk of the human infection is very much greater through the *handling* of measly pork than through the *ingestion* of it. Leuckart bears out this point and mentions that he found infestation with *T. solium* far more frequently in women than in men, and especially among cooks and kitchenmaids who handled pork, and when found in males, most frequently in butchers. In our Native Reserves the older men and the women most frequently handle pork carcasses. Owing to the sticky nature of pig measles and the greasiness of the lard, viable *cysticerci* can readily be conveyed by the hands to the mouths of such carcass-dressers. The young men, from whom mine workers are recruited, eat well-cooked measly pork in their Native Reserves, but the handling and preparation of the raw pork is mainly done by their women-folk. On one occasion the writer could have been subject to tapeworm infection, when he found a viable *Cysticercus cellulosae* on the mouthpiece of his cigarette, after having minutely dissected a measly pig carcass for observation purposes. I do not suggest that the incidence of *T. solium* in South Africa is as great, or nearly so, as that of *T. saginata*, but I do believe that in actual fact the incidence of infection is not truly reflected in observations on native mine workers. A high percentage of infection with *T. solium* should be observed if systematic faecal examinations could be made of representative colonies of natives, including those who most commonly handle measly pork in the Reserves, namely the women. During 1936 I had occasion to apply to Dr. Viviers, District Surgeon, Vereeniging, for some information regarding the origin of a case of cerebral cysticercosis in a native who died in the Bloemfontein Mental Hospital. The patient originated from the Vereeniging District. (See Case History Native Lucas Mpake in a subsequent portion of this Part.) Dr. Viviers replied, *inter alia* (letter dated 10.12.36): "I know that at least 25 per cent. of the natives in this (Vereeniging) district are infected with *Taenia solium*. In compounls, of which there are many in this area, the incidence of infection is higher." Upon first sight the remarks of Dr. Viviers may appear to be a somewhat exaggerated "guess in the dark", but to those who know the South African native and his habits, it will be clear that no gross exaggeration is presented. During 1932, I had occasion to satisfy my curiosity as to the extent of infection of humans with tapeworm in one of the Native Reserves. On that occasion, along with an Extension Officer attached to the Native Affairs Department, I visited the *stad* of the Chief of the Moiloa Native Reserve in the Marico District (N.-W. Transvaal.) At a meeting of about 4,000 natives, which I addressed on the subject of anthrax control, I was eventually questioned by one of the Headmen on the reason why so many of their cattle and pigs are condemned for measles, when sent to the Johannesburg and Pretoria abattoirs. After a brief outline of the life-histories of the two parasites I gave the opinion that many of the natives present must have been infected with tapeworm, and bluntly, in order to appease my curiosity, I asked those infected to show their hands. At first, apparent coyness caused only a few to admit, but after my assurance that there was really nothing to be ashamed of and that I would suggest a simple line of treatment to them, what I estimated to be between 15 and 30 per cent. of those present caused a mass of hands to be shown. It is, therefore, suggested that but a few of the actual carriers of tapeworm infection present themselves for treatment at various hospitals, such as those institutions for natives in Bechuanaland and Basutoland. As long as no great discomfort and physical pain due to the infection may be experienced by native carriers, these will not come to European physicians for treatment, and some, undoubtedly, are treated by their native " doctors ". The native's mentality and his suspicion of European interference with his ailments are amusingly reflected in a letter dated 14.5.33 from Dr. J. S. Armstrong, Medical Officer, Singida, Tanganyika, to Capt, H. J. Lowe, M.R.C.V.S., Veterinary Officer, Mpwapwa: " Upon receiving your letter I made an attempt to induce out-patients at this hospital to attend for the examination of their stools, but I regret that the only result was that all ran away before the treatment (of their other ailments) was completed."

In areas where proper meat inspection is not undertaken, the risk of infection to humans is great, and the incidence correspondingly high. Cawston (1935) related an astonishing fact. He wrote: " Seven years ago (therefore about 1928.—N.F.V.) some 30 per cent. of school children attending the clinic of the Potchefstroom Health Committee were found to be suffering from tapeworm infection, and this was used as evidence of the need for the establishment of an approved abattoir ". The Potchefstroom experience should surely have been a warning to smaller communities, especially those close to Native Reserves.

Unfortunately no statistics of the incidence of tapeworm infection in school children in the Union are kept. According to Dr. H. Maugham-Brown, Medical Inspector of Schools, Cape Province (letter dated 28.12.36), the incidence of infection in school children " seems to be higher in the Eastern Province than in the rest, more particularly in the areas which obtain their cattle from grazing areas occupied by natives ". The Chief Medical Inspector of Schools, Transvaal, writes (8.1.37):— " During the routine medical inspections the School Medical Officers do not examine all children on the presence of any intestinal parasites, on account of lack of time and facilities. Any statistics that may have been compiled out of facts obtained from medical inspections are very inaccurate and are only obtained from (*a*) direct information from the child, without being questioned in this direction, and (*b*) information obtained from the child on account of being questioned in this direction. Our experience, however, is that the incidence of tapeworm in European school children is fairly high in the rural areas of the Transvaal, and especially in the so-called Bushveld Areas (e.g. Marico, Zwartruggens, Rustenburg, Waterberg, Lydenburg, etc.) and this, presumably, is closely connected with the fact that in these areas cattle farming is the main occupation, and that in addition to this the native population is probably heavily infected with tapeworms. Roughly stated, the incidence of tapeworm in European school children in rural areas of the Transvaal ranges from a fraction of a percentage to as high as 20 to 25 per cent.

On the strength of this collection of evidence, it is clear that the problem of taeniasis infection in rural and native South Africa is most important, and warrants the scientific investigation of the medical profession.

After having considered these various facts, we are still no further in our decision as to whether *Taenia solium* or *Taenia saginata* is the more important parasite in the field of public health. If we accept the frequency of occurence of each individual species, as gleaned from actual observations, which, admittedly, reflect an incomplete survey, then *Taenia saginata* must be considered of prior importance. If, on the other hand, we must accept available medical evidence as to which species is the more damaging to the host, and is responsible for the more grave sequelae, then one must surmise that *Taenia solium* is the more important.

We have already mentioned the fact that *Taenia saginata* is more frequently the more " solitary " species in the host. (See Parts I and II.) It stands to reason, that the ingestion of an insufficiently cooked, heavily infested piece of pork, may cause the development of a great number of *Taeniae solium*. The chances of gross infestation through the ingestion of measly beef are less, owing to the general lighter infestation of the bovine. An exceptional infestation of a native was mentioned, however, by Watkins-Pitchford (1923), who stated that on one occasion as many as twenty specimens of *T. saginata* were recovered on autopsy, from the intestines of a single native.

THE EFFECT OF TAENIASIS INFECTION ON THE HUMAN HOST.

In the healthy adult an ordinary single infection with either parasite may not have very severe clinical effects on the patient. Yoshino (1934), who deliberately infected himself for experimental purposes with *Taenia solium*, found that the presence of a few adults in a patient would cause only slight gastro-intestinal derangement, which was usually more manifest in the early stages of infection. So little physical discomfort is felt by some of the more primitive peoples, e.g. the Abyssinians (Schimper, quoted by Leuckart) to infection with *Taenia saginata*, that these people maintain " that without this guest they would be unhealthy, and that they would suffer especially from constipation ". According to Leuckart, intestinal irritation and nervous derangement in the host is much less frequent in *Taenia solium* than in *Taenia saginata*, but on the other hand, the presence of the hooklets on the head of the former sometimes causes injuries to the intestinal mucosa. Attachment of both species occurs in the small intestine. " When in possession of its full vital powers, the worm hangs so firmly that it is necessary to pull and bend it before it will quit its hold. And even after it has done so, it will attach itself again in a moment, if the head succeed in catching hold of a portion of the intestine." (Leuckart.)

According to Braun-Seifert (1923), the infection may cause the following derangements in man:—

1. *Absorption of Nourishment*: The loss of nourishment on the part of the host is usually compensated by the eating of larger amounts of food, owing to the abnormal appetite the patient develops (*Heiszhunger*).
2. *Digestive derangements*: Frequently diarrhoea, followed by constipation etc.—frequently flatulus, tympanites, sometimes spasmodic colicky pains, and sometimes a "pressure" in the abdominal region.
3. *Nervous derangements.*
4. In weaker individuals *anaemia* may easily follow.

Sequelae.

Braun-Seifert (1923) refers to Spengler, who performed an operation for appendicitis on a 29 years old woman, and found a live proglottis lodged in the appendix. According to Spengler, this proves that the presence of a foreign body will cause, through friction, the symptoms of appendicitis simplex. Martin, Pollag, Retzlaeff and Westermann found similar causes of appendicitis. (Braun-Seifert.)

Altenkamp (1935) recorded a case of acute appendicitis, in which the presence of a portion of the strobila of a *Taenia* was the cause. So also did Pytel (1935) refer to a case in which tapeworm infestation was the cause of appendicitis.

Farzane and Ibragimov (1935) found that ileus of the intestine had been caused by a conglomeration of *Taenia solium* segments.

Leuckart asserts that it is quite conceivable that the powerful contractions of *Taenia saginata* have an influence on the condition of the intestine. The projecting borders of the joints thus rub in a file-like manner over the villi and easily produce a congested state, which lasts a longer or shorter time according to the circumstances, and gives rise to many disease symptoms. If the disease continue long, the nutrition suffers. From this there often arises a condition which has a certain resemblance to anaemia, and which especially exhibits the many neurotic symptoms of this disease. "Singing in the ears, hallucinations, giddiness, fainting, pains in the joints, epilepsy, chorea and even mental diseases, have all been observed to be caused by the tapeworm, and not infrequently to disappear on the removal of the latter." (Leuckart.)

Burnet (1919) placed on record three cases of chorea, which had their origin in the presence of tapeworm, and were cured when the worm was expelled; he pointed out, however, that a rheumatic tendency might have been a predisposing cause.

Very interesting work on the subject of psychosis due to tapeworm infestation was done in South Africa a few years ago, by Dr. A. S. van Coller, who was formerly on the staff of the Bloemfontein Mental Hospital, and is now Physician Superintendent of the Mental Hospital, Grahamstown. Dr. van Coller very kindly supplied me with a memorandum of his research, and has given me permission to quote his hitherto unpublished findings.

Out of 450 cases, all suffering from psychosis, he found two groups, viz.

" a " Toxic Group (180).

" b " Exhaustion Group (130).

Both these groups he considered were directly due to tapeworm of the intestines. After treatment the " a " group responded almost immediately—that is, they recovered within three months. The " b " group was much slower—here recovery was slow—blood examination revealed a secondary anaemia in practically all cases. It usually took from three to twelve months for a recovery. The anaemia had disappeared by the time recovery was established. So in this group he reckoned the tapeworm had been present for such a term as to produce anaemia. The toxaemia, plus anaemia eventually caused a psychosis. The balance, that is 136 cases turned out to be a mixture of classical types, mostly Dementia Praecox. (The remaining four cases were of Cysticercosis of the brain, of which three died from epileptiform psychosis—see later.)

Dr. van Coller was of opinion that in these classical types (Dementia Praecox, etc.) the tapeworm acted mainly as a precipitating factor, not causal.

Dr. H. Egerton Brown of Pietermaritzburg, and formerly of the Union Mental Hospital Service, informed me (letter dated 25.1.37) that he was convinced that a certain number of cases of acute excitement (mania) among native admissions to the Mental Hospital was due to an absorption of a toxin secreted by the living *Taenia*, and he made it a routine treatment to try and expel the parasite in all cases of this nature. Dr. Egerton Brown kindly supplied a record of 207 positive cases of tapeworm infestation. Of this number 139 recovered after treatment and were discharged. Sixty-eight cases were relieved, or not improved. Of the recovered cases, diagnosis was Hebephrenia or Simple Dementia Praecox and Toxic cases. The relieved or not improved cases all showed an improvement for some time after treatment. " On retreatment, the same thing happened. Three cases of epilepsy due to tapeworm infection were discharged as recovered after treatment. No fits for many months after treatment, they have not been readmitted."

The findings of Doctors van Coller and Egerton Brown have been quoted to illustrate the occasional dangerous sequelae of tapeworm infection, and have, of course, a special South African interest.

Both species of human *Taenia* may occasionally show amazing tenacity of life. " These tapeworms grow to a length of twenty to thirty feet and can live for 12 to 20 years, or even longer."

(Mönnig, 1936.) Franke (1931) referred to the tenacity of *T. saginata.* Some cases he knew of had harboured the parasite from 15 to 19 years, and in one case the patient required six vermicides before the tapeworm was eventually expelled. Leuckart quotes Cobbold, who had cases who evacuated proglottides daily for 11 years, and Wawruch, who mentioned several cases which lasted from twenty to twenty-five years, and in one case he even mentioned thirty-five years. " Of course, it is doubtful whether this is always the effect of the same tapeworm ". (Leuckart.) Leuckart mentions that occasionally after death of the tapeworm, and instead of resultant expulsion, mummification of *Taeniae may* occur within the host's intestine. Such mummified specimens were found by Cobbold and by Küchenmeister.

Conditions which were caused by what Shahan (1932) referred to as " migratory *taeniae* " have occasionally been recorded. A soldier in the Egyptian Army, with a history of occasional attacks of suffocation, died in hospital under Shahan's treatment, from a distressing dyspnoea. On post-mortem examination a tapeworm was found lodged in the larynx and upper part of the trachea. Shahan mentions a case in the literature in which incision of the drum of the ear for severe earache was followed by the passage of a tapeworm from the middle ear and eustachian tube. Cases are known in which the whole or portions of a tapeworm have been vomited. Lavalette (Leuckart) reported the case of a pregnant woman who expelled the proglottides singly through the mouth. Leuckart refers to cases in which proglottides, or even the entire strobilae had passed through fistulae of the bowels into the abdominal cavity. " Especially interesting in this connection is a case mentioned by Herz, in which the tapeworm issued through the navel, without, however, bringing any of the contents of the intestine with it, so that the patient could be dismissed as cured a few days after the exit of the worm ". Leuckart also records rare cases in which the tapeworm was expelled through the urethra. " In such cases, even when the ordinary signs of vesico-rectal fistula are wanting, it is evident that the worm can only have reached the urinary apparatus from the intestine. In one of three cases mentioned by Davaine, the tapeworm remained a year in the bladder, and expelled single proglottides at intervals of about eight days, until it was killed by an injected anthelminthic and then expelled at once. We need hardly add that expulsion of proglottides through the urethra is accompanied by violent and painful disorders, and that the above-mentioned cases interfere in many ways with the health of the host."

To summarize, we might mention that:—

(1) Simple infestations with either *Taenia* have not, as a rule, any serious damaging effect on the human hosts, but

(2) Anaemia, with resultant debility may follow.

(3) Frequently both tapeworms may show amazing tenacity of life, and may remain alive and actively eject mature proglottides for many years.

(4) Both species may be responsible for very serious sequelae, among which have been recorded:—

(*a*) Digestive derangements and pathological conditions of the intestinal tract—ileus of the bowel, appendicitis, intestinal fistulae, which may cause migration of the parasite to the uro-genital tract.

(*b*) Nervous and mental derangements—e.g. chorea, psychosis with dementia praecox, epilepsy due to *Taenia* infection.

(*c*) " Migratory *taeniae* " may lodge in the respiratory tract (usually in the upper part of the trachea and the larynx), and in rare cases segments may be found in the acoustic and olfactory regions, e.g. in the eustachian tubes.

THE INFESTATION OF THE HUMAN SUBJECT WITH CYSTICERCI.

Of perhaps greater importance from a clinical point of view than infection with the adult *Taeniae*, is the infestation of the human subject with *Cysticercus cellulosae* and *Cysticercus bovis*, which, according to Broughton-Alcock, Stephenson and Worster-Drought (1928) has been known since 1558. According to consensus of opinion, the former parasite is far more frequently met with in the human subject, and many authorities doubt whether actual cases of bovine cysticercosis have been encountered in man. Leuckart accepts, with a great deal of reservation, the probability of human infection with *cysticercosis bovis*. Mönnig refers to the possibility of such infection, but qualifies this with the reminder that the *C. cellulosae* is by far the more frequent parasite (1934). Von Ostertag (1934) states that *C. bovis* " has never been definitely found in man ". It is, therefore, almost certain that by far the large bulk of cases of human cysticercosis (to be clearly distinguished from human echinococcis, or so-called hydatid disease) is due to infection with *C. cellulosae*.

In every case of human cysticercosis, the victim of infection has undoubtedly been directly or indirectly in contact with a *Taenia* carrier. Such infection may result from the following:—

1. *Auto-infection*: According to case histories, and also to the opinions of many authorities, it is less frequent that cases of human cysticercosis have been met with, in which on post-mortem or other examination evidence of *Taenia* infection was found. When such cases occur, auto-infection may result from:—

(i) Anti-peristalsis, in which reverse movements of the intestinal contents lodge in the stomach, and in such movements carry ripe proglottides of the *Taenia solium* with them. The ova are liberated and human infection follows on similar lines to that of the pig. (See Part III.)

(ii) Conveyance of ova on the fingers, or under the finger-nails into the mouth, by a *Taenia* carrier. In such cases the tapeworm carrier, most commonly of *T. solium*, will infect himself with *C. cellulosae*. Vosgien

(1910-11) mentions that out of 579 cases of human cysticercosis observed by Auscher, 62 were infected with *Taenia solium*.

2. *Infestation due to ingestion of* Taenia *ova by a carrier and contamination of food-stuffs*: This mode of infection is probably responsible for the great bulk of cases of human cysticercosis. Chin (1933) was of opinion that the ingestion of insufficiently cooked vegetables was the greatest source of infection in China. A similar opinion was given by Vosgien (1910-11). Colonel F. P. Mackie (1934), in a discussion on MacArthur's paper on "Cysticercosis as seen in the British Army", comments on the fact that in India only the lowest caste inhabitants eat or touch pork, and it is probably they, in preparing and handling soldiers' food directly, or in eating houses in bazaars, who, as *Taenia* carriers, were responsible for the infection of the number of cases cited by MacArthur.

The use of human excretum as fertilizer for vegetable gardens is a grave source of infestation of cysticercosis to the human subject. Such vegetables as lettuce, parsley, celery and water-cress, the leaves or stems of which are generally eaten in a raw state, are positively dangerous if human excretum has in any way come in contact with them. It is highly probable that the majority of cases of human cysticercosis found in South Africa, especially in Europeans, originate through accidental infection through the ingestion of ova in the food. We run a grave risk of infection in South Africa through our close contact and association with our native population. Garth (1923) stressed the potential danger in a household of a *T. solium* carrier, whose contact with the rest of the family could cause the ingestion of *Taenia* ova by them.

It is a remarkable fact that most of the case histories in British literature have reference to infection in India and elsewhere in the Orient, and nearly all refer to subjects who served in the Army or Navy. [Reference articles by MacArthur (1934), Dixon and Smithers (1935), Dudley (1934), Dick (1936), Holmes (1934), Lindeman and Lyburn (1935), Marsh (1934), Perry (1936), Broughton-Alcock, Stephenson and Worster-Drought (1928), Priest (1926), and Roth (1926).]

The incidence of *Taeniasis solium* is not known in India and China, but, according to MacArthur and others, it can only occur among the very lowest caste in India, and, as has been mentioned, according to Mills (1923 and 1924), Gear and Pedersen (1934) and Feng (1934), the recorded incidence of *T. solium* is very low in China. In our survey of the incidence of infection in pigs, it was also recorded that, from abattoir observation, the incidence of porcine *C. cellulosae* was almost negligible in that country. In South Africa, in many parts, the incidence of porcine cysticercosis is very high. Although we have no data to prove our surmisal, except the opinions of some medical observers, it is, nevertheless, almost undeniable that *Taenia solium* must be a very frequent parasite among our native population. It is they, who generally handle our food, and we are thus seriously exposed to a far more dangerous infection than we would ordinarily acquire through the eating of viable measly pork or beef.

It has been suggested by some writers (Vosgien, 1910-11) that heredity may also be a factor of transmission of infection. This factor we mentioned in dealing with infestation of the pig and the bovine.

Volovatz (1902), according to Vosgien, " in his highly documentary thesis draws attention to the fact that *cysticerci* have been found in placentae; this might explain the origin of this entozoon in new-born infants ".

Breast-fed, suckling infants, however, must run the smallest risk of infection, since their only diet is their mothers' milk. Heller (1874), according to Vosgien, mentions, however, a case of a 6 months old child who had a *cysticercus* in the mesentery. Vosgien also quotes Virchow (1877) who found this larva in a 9 months old child; Karewski (1877), who found *cysticerci* in a breast-fed child whose mother carried *Taenia solium*.

It would appear that in the majority of cases of human cysticercosis, the onset of infection occurs between the ages of 20 to 40 years. It is, thus, usually a disease of adults. According to Vosgien, out of a total of 478 cases observed, 206 occurred between those ages. Dudley (1934) described a case of epilepsy in a sailor, and based his diagnosis of *C. cellulosae* as being the cause of the disability on: Age of onset, 40 years; place of origin, China; infestation with an adult *Taenia solium;* plus X-Ray appearance of calcified cysts.

Locations of Cysticerci in Man.

The brain is commonly held to be the most frequent site for *Cysticercus cellulosae* in man, but this may merely be because cerebral symptoms, when they show themselves, are more marked than muscular symptoms (Chizzola, 1933). Similarly, MacArthur (1934) explains that ocular and orbital cysticercosis causes outward signs which can hardly be unobserved, whereas intramuscularly they may exist for years without the patient or his associates observing them. None of a large number of calcified cysts in the arms and legs of a case described by Chizzola, and demonstrated radiographically, was palpable. This rather suggests that *cysticerci* are usually situated intramuscularly, rather than subcutaneously.

McCrae (1931) quotes Stiles, who compiled statistics of the locations of infections as follows:—

In 155 cases, the brain was involved in 117 cases; the muscles in 32; the heart in 9; the subcutaneous tissues in 5 and the liver in 2.

Vosgien found the predilection sites in man to be:—

	Per Cent.
Eyes and adjoining structures	46.
Brain and Nervous System	40·9.
Skin and Cellular Tissues	6·32.
Muscles	3·7.
Other organs	3·2.

Cysticercosis of the Muscles, Skin and Intracellular Tissues and Other Organs: A Review of a few Recent Case Histories.

Priest (1926) mentions a case in which five years after enlistment in the Army a private became sick, showing abdominal pains and vomition. His liver was found to be enlarged. He then developed pain and swelling in the calf muscles, which on examination were found to be nodular. A few months later he had a " fit ". He showed a chain of nodules on the forehead and muscles. There was no evidence of the patient having harboured a tapeworm, so that he must have become infected through ingestion of extraneous segments of ova.

Radiology has been instrumental in the discovery of quite a number of cases of cysticercosis in man, especially when calcified cysts have been located in the limbs. Capua (1932), having detected calcified *C. cellulosae* in the musculature radioscopically, suggested that this method of diagnosis should be employed in all suspicious cases.

Roth (1926) describes a case of cysticercosis in a man 44 years of age, a hairdresser. He complained of pains in the left knee-joint, of five years' duration. For ten years the patient had suffered from epileptiform " fits ". He served in India from 1908 till 1911. Radiographs of the left knee were taken, which disclosed some 80 calcified cysts. It was then decided to take radiographs of the rest of the body, and these showed calcifications in the extremities down to the ankles and wrists. The pectoral muscles showed numerous cysts. Three bodies were seen to be lying in the pia mater of the brain. Similar cases were described by Gavazini (1934) and Casuccio (1933). Streignart (1933) demonstrated calcified *cysticerci* in the muscles of a peasant's leg which was X-rayed for fracture. Disseminated calcified *cysticerci* were found in a case by Kremser (1934), also by Grado (1935),and were demonstrated by X-Ray. Brailsford (1926) demonstrated *cysticerci* in the thigh of a patient radioscopically. Chin (1935) described thirteen cases in Peking, which showed " nodules " or tumors under the skin, and were found to be due to *C. cellulosae.*

According to MacArthur (1934) cysts may be detected in the muscles or subcutaneous tissues of any part of the body—the head and the face, including the eyelids and lips, trunk and limbs, but rarely in the hands and feet. They are found more commonly in the upper half of the body, not because the parasites are more numerous here, but because of the better cover afforded by the larger masses of muscles in the lower half. Among other unusual locations of *C. cellulosae* is the mammary gland. Such a case was described by Stumpf (1915).

In 26 cases of cysticercosis in the viscera and " other organs " mentioned by Vosgien, *Cysticerci* were found in the heart in 10 cases glands in 6 cases; digestive organs in 3 cases; lungs in 2 cases; and in the mouth in 1 case.

Cysticercosis of the Eyeball and its Annexes.

Vosgien (1910-11) mentions 372 cases of ocular (or related) cysticercosis out of 807 observations. Of these cases:—

The retina	was affected	120	times.
The vitreous	,, ,,	112	,,
Subconjunctiva... ...	,, ,,	84	,,
Anterior Chamber ..	,, ,,	26	,,
The orbit	,, ,,	19	,,
The iris	,, ,,	7	,,
The crystalline ...	,, ,,	2	,,
The cornea	,, ,,	2	,,

Vosgien quotes Hirschberg (1892), who " made a remarkable observation, that the *Cysticercus* apparently had a different predilection in various territories ". " It is difficult to explain ", states Hirschberg, " but the predilection of the *Cysticercus* in France is for the conjunctiva, in England for the posterior chamber, and in Northern Germany for the various parts in front of the eyeball ". One wonders, however, whether coincidence may not have been the more important factor which caused Hirschberg to have made this peculiar observation. Since von Graefe (1866) demonstrated the presence of the *Cysticercus* in the vitreous humour, many cases have been placed on record, and it is a condition which is easily recognized.

According to Burdon-Cooper (1921), three different species of tapeworm larvae occur in the eye, namely that of *Taenia solium*, that of *Taenia echinococcus* and the *Bothriocephaloid* tapeworms, but the first named is by far the most common.

It has been found possible to remove *Cysticerci* successfully in eye affections. Two such cases were described by Gomes (1919). In the one case the *Cysticercus* was located in the anterior chamber, and was removed without difficulty; the other was removed from the vitreous chamber by incising the sclerotic, the procedure being guided by ophthalmoscopic examination.

Pavia and Durando (1933) described a case of *C. cellulosae* which developed behind the retina of the right eye in a woman, 31 years old. The changes in the vision of the patient, as the cyst grew, were traced by the authors.

Among some interesting cases recorded of *Cysticercus* of the vitreous, mention may be made of a case described by Schweinitz and Wiener (1919). In this case the left eye was involved and was blind five months before the patient came under examination—blurred vision being noted ten months previously. There were no gross changes in the iris, but a few punctate deposits on Descemet's membrane and a few spots on the anterior capsule of the lens. The vitreous was cloudy with a few fixed vitreous opacities. There was a grey reflex in the upper and inner quadrant, " and a large globular mass in the central field of the vitreous, well in advance of the maculose region; the outline of this was regular, the border translucent and from the lower edge protruded a tubular extension, transversely wrinkled, which terminated beyond a constricted neck in a head showing two bright dots—the position of the hooklets. Peristaltic movements and movements of the head were very active at times." The diagnosis of *Cysticercus* of the vitreous was readily made. The general examination of the patient revealed little except that the stools contained ova but no segments of the worm. Operation was undertaken and the cyst removed, when it ruptured promptly.

Kress (1924) gives a most graphic and interesting description of the development of a *Cysticercus* in the vitreous, which occurred in a woman of 26. In the author's own words it is summarized somewhat as follows. There was at the onset, what seemed to be a detachment of the retina, which, under dilatation at a later date proved to be a bluish-white cyst, which was of a perfect spherical shape and moved slowly in the vitreous, with movements of the patient's head. This bluish-white cyst had, practically at all times, an orange or orange-red halo at its periphery, shading off somewhat as do the colours of the spectrum. Later the greyish-white head and neck of the parasite put in an appearance, at about " 5 o'clock meridian ", and this neck could change its shape and become thicker through contraction, and it could bend itself and twist on itself, and at times invaginate or probably contract within the cyst until practically nothing but a slit was seen at its former site. The activity of the head and neck movements and of the suctorial and snout or rostellum areas, as well as the undulating movement of the vesicle proper, could be seen ophthalmoscopically. The eye had to be excised. Casânovas (1933) described a case of atrophy of the bulb of the eyeball, as a result of intraocular cysticercosis. Chica (1925) reported a very similar case to that described by Kress. He reminded us that this was the third case he had seen in Bucharest.

According to Feng (1934), less than 20 cases of ocular cysticercosis have been reported from China. Rao (1935) recalls that Wright of the Madras Ophthalmic Hospital has found that 3 per cent. to 6 per cent. of cases in that hospital had ocular cysticercosis. In consideration of this relatively high incidence of ocular cysticercosis, it must reasonably be presumed that the incidence of *T. solium* must also be relatively high in parts of India.

Cerebral Cysticercosis.

Cysticerci cellulosae may be present in the human brain, and also in other organs and the musculature for many years before clinical disorders become manifest. Although, in a case described by Billello (1934), more than 1,000 *cysticerci* were present in the cortex of the brain and the fourth ventricle, hardly any symptoms were present before death, and cysticercosis was not suspected. Occasionally records occur of admission to hospital for headache and unidentified fever, or for myalgia or rheumatic pains, " but these latter are usually of a degree so indefinite as not to impress the patient's memory." (MacArthur, 1934.)

In the brain, the *Cysticercus* becomes enclosed by a wall of sclerosed neuroglia, corresponding to the fibrous capsule found in extracranial tissues. Small round cells and a few plasma cells are present between the delimiting neuroglia and the surrounding normal brain tissue. " Unless the parasites have invaded the brain in overwhelming numbers, or have lodged in some particularly responsive centre, they cause little nervous disturbance while in their relatively quiescent stage, otherwise it seems impossible that anyone could survive for years—as we know to be a fact—with 200 *cysticerci* present in the brain." (MacArthur, 1934.) Surrounding the dead and disintegrating *Cysticercus*, the tissues undergo active degeneration (MacArthur, 1934; Heilmann, 1932). MacArthur believes that *Cysticerci* while alive usually enjoy a relative tolerance on the part of the host, but after their death they act as foreign irritants and bring about the degenerative changes. The degenerating tissues may be visible around the *Cysticercus* as a discoloured ring, according to MacArthur, perhaps 3 mm. or thereabouts in depth, shading off into the normal brain tissue. *Cysticerci* may be present in the brain or in the body muscles for many years before cerebral symptoms become evident. MacArthur refers to cases ranging from six to eleven years, and, according to MacArthur, when brain symptoms develop, they are subject to periods of exacerbation, followed by intervals of relative or absolute quietude, and the character of the symptoms may vary so markedly that an individual patient seen at intervals by different observers has been diagnosed as delusional insanity, disseminated sclerosis and cerebral tumour. Such cases of cerebral cysticercosis simulating clinical aspects of brain tumours, were also described by von Lehoczky (1933). Antonow (1932) stated that young *Cysticerci*, living at the time of the host's death, are enclosed in a thin capsule composed of an outer layer of granulation tiusse, containing giant cells. Around older *Cysticerci*, which have died, the capsule is thick and has in addition an inner connective tissue layer, giant cells being here and not in the granulation layer. As a final stage Antonow described a single thick layer of hyaline connective tissue. MacArthur has found that degeneration of human *Cysticerci cellulosae* has occurred somewhat in the reverse order from that described in the literature in respect of pig measles. Instead of degeneration commencing and progressing from the vesicle, he has noticed in some newly degenerated excised cysts that calcification commences in the scolex, and the bladder, with its fluid contents, has remained unchanged. The calcified scolex may lie " quite free " in this. According to MacArthur the cyst wall collapses after this, causing escape or absorption of the fluid, and leaving merely the solid calcified scolex. According to MacArthur calcification of cerebral *Cysticerci* takes many years to occur. He refers to the case of one soldier who was operated on eleven years after the onset of " fits ". Several cysts removed from the cerebral cortex showed no signs of calcareous change, although the cysts in the muscles had been calcified for three years and some for five years.

The location of the parasites in human cerebral cysticercosis may be very variable. According to Heilmann (1932), *Cysticerci* may be free in the ventricles, blocking Magendie's foramen or the Sylvian aqueduct. In one case Heilmann found Monro's foramen occluded.

Of the nervous manifestations, by far the most common is epilepsy. The attacks may resemble those of *petit mal*, or may be Jacksonian in type, with, or without, loss of consciousness. MacArthur has proved that *Cysticercus cellulosae* in the brain is a frequent cause of epilepsy in British soldiers who have served in India. Within about eighten months, just prior to 1934, MacArthur met with sixty such cases at the Millbank Military Hospital. Dixon and Smithers (1935) mention that of 258 suspicious cases examined at the Queen Alexandra Military Hospital, 79 have been proved to be due to cysticercosis, and 40 were doubtful. Broughton-Alcock, Stephenson and Worster-Drought (1928) described a case of a young soldier who had died, aged 28, after having served in India. For several years after he enlisted, the patient suffered from epilepsy. On post-mortem about 100 cysts were found in the half-brain.

Dick (1936) describes a case of a man 50 years of age, who had served in India 12 to 14 years ago. Between 1923 and 1933 he was engaged in a shipyard. In 1933, he began to suffer from epileptiform seizures and was later admited to an epileptic colony, where he remained until he died after about a year. He was subject to fits of depression, occasionally with confusion, and at one time he had an outburst of religious mania. In 1935, he began to suffer from cerebral vomiting, which increased in frequency and a severe and persistent headache developed over the occipital region. After death, autopsy showed numerous cysts in various parts of the brain.

Ramond (1933) described a rather unusual case in which a 35 years old woman showed symptoms of what appeared to be Jacksonian epilepsy, the cause of which remained obscure for some time. Eventually the cause was traced to a multiple infection with *Cysticercus cellulosae*. Flossbach (1932) mentions a case in a 43 years old woman, who had tapeworm in 1912. Twenty years later she had epileptiform convulsions due to cerebral cysticercosis. Lindeman and Lyburn (1935) had two cases of epilepsy in the British Army, due to cysticercosis, and they suggested several others. Similar cases in the British Indian Army were recorded by Holmes (1934) and by Perry (1936).

MacArthur mentions that the epileptiform seizures may at first be slight and incomplete, but after a year or so major seizures in rapid sequence may succeed. Frequently the "fits" may commence a long time after the presence of cysts are detected.

Other nervous derangements which may manifest themselves in cases of cerebral cysticercosis are acute encephalitis causing rapid death, melancholia, acute mania, delusional insanity and dementia praecox. A case of basilary and spinal meningitis due to *C. cellulosae* in a 61 years old patient, who had been under observation for some months, was described by Guillain, Bertrand and Thurel (1933). Diagnosis was confirmed by post-mortem examination. A similar case was reported by Liesch and Patrassi (1934).

MacArthur suggests that in the established disease, when the embryos have been "walled off" there is no diagnostic help to be gained from blood counts, but when the embryos are still active in the body, no doubt an eosinophilia results. Presumably for the same reason, the complement-fixation and skin tests—which are group reactions—have not the high degree of success of the corresponding tests in schistosomiasis, filariasis and hydatid disease.

Rizzo (1932) diagnosed a case of human cerebral cysticercosis during life, largely upon the finding of an eosinophilia in the cerebro—spinal fluid, accompanied by a negative Wassermann reaction—the case did not harbour the adult *T. solium* and infection was confined to the central nervous system.

Fairley, according to MacArthur (1933), obtained a positive complement fixation in 5 cases out of 9 tested.

" Careful enquiries into the value of an eosinophilia, as suggesting the presence of *Cysticerci* has shown that the blood-count is not an entirely reliable guide. During the acute stage of infection with either *Taenia* or *Schistosoma*, most persons develop an evident eosinophilia. The complement-fixation test has proved of value in certain cases, but even this needs to be confirmed by other tests." (Cawston, 1935.) In infections with the adult tapeworm, however, Kawanishi (1932) found marked leucocytosis in four persons intentionally infected with *T. solium per os*. Eosinophilia was only 15 per cent. Neutrophiles showed an increase and lymphocytes a decrease.

As regards treatment, luminal and the bromides are sometimes helpful in controlling fits, but no medicinal treatment has as yet had any curative effect. MacArthur is of opinion that any drug which might be found to be lethal to live *Cysticerci* may be equally damaging to the tissues of the host. On account of the usual large number of cysts in the brain, surgical treatment cannot be resorted to.

SOME RECORDS OF HUMAN CYSTICERCOSIS IN SOUTH AFRICA.

It will be noticed from the subjoined number of case histories supplied by various Mental Hospitals, that quite a number of cases of human cysticercosis has been encountered in South Africa. Nevertheless, literature is singularly silent on the subject of cysticercosis in humans in South Africa, and very little has been published in South African and Overseas journals by our medical observers.

Cawston (1935) refers to a case of the late Dr. Barry, in which the brain of an adult native who had died after being struck on the head, had revealed numerous *Cysticerci*. Apparently no ante-mortem symptoms were observed by those who had come in contact with the native.

Pirie and Ray (1920) showed a case of generalized cysticercosis in a native. There was a great number of *cysticerci* in the muscles, both of the trunk and the limbs, also in the diaphragm. There were moderate numbers of *cysticerci* in the brain, the heart and over the pleurae. There was no history of any illness in this native.

Strachan (1926) described the brain of a native male aged 56 years. This native was picked up in the street in a delirious condition, with a temperature of 101° F. On post-mortem examination his brain was found to be riddled with *Cysticerci cellulosae*. Strachan mentioned that he had found four cases in two years, and described a second case of a native with *C. cellulosae* in the heart, without brain lesions.

Fischer (1929) recorded, in his paper on "Autopsies on Native Mine workers", cases of *Cysticercus cellulosae* accidently found in the brains of three patients, who died of other diseases. In two cases a number of cysts the size of a pea was found on the surface of the frontal convolution; they could easily be squeezed out, without leaving any visible damage to the brain substance. In the third case a single *Cysticercus* was situated in the left lateral ventricle.

Hospital Records.

Pretoria Mental Hospital.

The data of the undermentioned case histories were very kindly supplied by Dr. I. R. Vermooten, Assistant Physician Superintendent. Dr. Vermooten was able to find the records of only five cases between the years 1908 and 1934.

1. Male Native. Age 33; Admitted 4.8.16.

   Diagnosis: Epilepsy.

   Had epileptic fits since admission—put into bed after a succession of fits and remained in a semi-comatose state for seven days. Died 15.9.19. *Autopsy:* *Cysticerci* scattered throughout the brain substance. Cysts in floor of the ventricles. Cause of Death: Lobar pneumonia, aggravated by *Cysticerci* found in the brain.

2. Female European, M.B. Age 71; Admitted 10.7.18.

   Diagnosis: Senile Dementia with Epilepsy.

   She was demented and chattered incoherently. Suffered from fits.

   29.7.20: Has had 15 very severe fits during the last two days, and is now only semi-conscious—muscles continue twitching—condition critical.

   3.8.20: Continues to have seizures—only semi-conscious.

   6.8.20: Died. *Autopsy*: Head—Calvaria shows very prominent ridges laterally over the temples. Skull is soft and in parts extremely thin, e.g. parietal eminences and just behind the coronal suture. Base of skull normal. Dura Mater: Thickening present. Adhesions marked. Lining shows some congestion. *Sub-dural space*—contains a large amount of C.S.F. *Pia-arachnoid*—Well marked opacity and milkiness. *Encephalon*—As there are numerous cysts in the brain, no dissection has been made and the brain has been put in its entirety into 10 per cent. formalin to harden. Head only examined. Cause of death: *Cysticercus* of the brain.

3. Female European, A.P., admitted 20.12.19. Age 75.

   Diagnosis: Senile Dementia.

   She was demented and very restless. According to the case sheets on 26th May, 1920, she had a severe seizure, lasting nearly five hours.

   10.6.20: Patient has been having frequent seizures during the past few days and is gradually becoming weaker.

   10.7.20: Died. *Autopsy*: Head—Calvaria much thickened. Almost ½ inch in frontal and occipital regions. *Dura mater*—Thickening very marked. Lining injected. Adhesions—yes, to bone. *Sub-dural space*—Contains a large amount of C.S.F. Quite abnormal. *Pia-arachnoid*—Opaque and thickened. Separates fairly easily from brain. *Vessels of Brain*—Injected and more evident than usual. Small patch of atheroma in basilar artery. *Encephalon*—Weight 1,200 grms. Weight of right hemisphere 485 grms. Weight of left hemisphere 495 grms. Over the surface of both hemispheres and under the pia-arachnoid there are numerous cysts containing a turbid fluid and varying in size from about ⅛ inch to ½ inch in diameter. The cysts are over the left frontal region where there is quite a depression made on the surface of the brain. They are also numerous on the right parietal region.

   Cause of Death: *Cysticercus* of the brain.

4. Male Native. J.M. Age 65. Admitted 8.10.21.

   Diagnosis: Senile Dementia.

   He is demented, unable to give an account of himself; speech indistinct; very deaf.

   15.12.21: He went into *status epilepticus* and had six fits. *Autopsy*: *Cysticerci* in large numbers all over the brain, Ventricles, Cerebellum and Fourth Ventricle—*Cysticerci* present.

   Cause of Death: *Cysticerci* of the Brain.

5. Male Native. Age 35. Admitted 31.10.25.

   Diagnosis: *Cysticercus* of brain and symptomatic epilepsy. He was dull, rarely spoke. Knew his name, but could give very little further information about himself. Paraplegia of right arm and leg. Had innumerable fits of a Jacksonian type. Hemiplegia aggravated after fits. Few small cysts in pectoralis major of left arm. Shortly before death he was dull and demented. Quite unable to do anything for himself. Died 26.7.30. *Autopsy*: Both hemispheres of cerebrum covered with cysts; many of the cysts lie loosely on the brain surface. Several small cysts scattered throughout pectoralis major and biceps muscles.

   Cause of Death: *Cysticercus cellulosae.*

Dr. Vermooten concludes: " I have no doubt that if a post-mortem had been done on all cases where the cause of death was ascribed to epilepsy, more cases of *Cysticercus* would have been discovered ".

Dr. H. C. Watson, Physician Superintendent of the Bloemfontein Mental Hospital, informs me (letter dated 4.2.37), that between the years 1909 and 1914 he saw at least half a dozen cases of cerebral cysticercosis in the Pretoria Mental Hospital.

Dr. F. D. Crosthwaite, Physician Superintendent of the Mental Hospital, Potchefstroom, and formerly of the Medical Staff of the Pretoria Mental Hospital, very kindly supplied the following data regarding *Cysticerci* in the brain in natives. Dr. Crosthwaite states (letter dated 15.1.37) that what they were investigating was the incidence amongst natives, who were epileptic, of parasitic cysts in the brain, and of the frequency amongst these natives of the cysts as a cause of epilepsy—the figures supplied, refer exclusively to natives certified as mentally disordered (epilepsy with psychosis) in the Mental Hospital, Pretoria.

Period: 1911-1918.—Number of Autopsies 288.

Of these 288 autopsies, 10 revealed the presence of cysts, situated as follows: 9 in brain; 1 in the heart. Of these 10, 7 were males, 3 were women.

Of the 7 males, 4 had epilepsy, 2 were dementia praecox (hebephrenia), 1 had syphilitic brain disease with *cysticerci* in the heart. Of the three females, all were dementia praecox.

During the same period, 1911-1918, there were 334 deaths. Of these 334 deaths, 44 were epileptics. Of the 44 epileptics, 34 were autopsied, 29 being males and 5 females. No cysts were found in the women's brains, but 4 of the men's brains had *Cysticerci*. Counting the cases autopsied only, i.e., 34, *Cysticerci* were found as the exciting cause of the epilepsy in 11·706 per cent. of the cases.

Potchefstroom Mental Institute.

Dr. Crosthwaite states (15.1.37) that during his five years at Potchefstroom, two things have struck him; the very low death rate, and the impossibility, almost, of getting permission to perform autopsies. They have over 100 Europeans who are epileptics, but epilepsy amongst the feeble-minded (low grades, imbeciles and idiots) is of very common occurrence, and is due, when it occurs, to the imperfect development of the nervous system, and its general inadequacy and instability, or to the presence of gross anatomical lesions.

Bloemfontein Mental Hospital.

Case History: Native Lucas Mpake.—Kindly supplied by Dr. G. de la Bat, who attended the patient.

Native Male from Vereeniging. Age 55 years. Admitted 6.6.36, died 16.6.36.

Cause of Death: "*Cysticerci* of *Taenia solium* in brain."

Mental State on Admission: Restless and unnaturally talkative. Second day after admission he developed clonic spasms involving musculature generally. He gradually became more dazed and confused. Twitchings became more marked on the right side, especially the facial muscles. Eyes became fixed and staring.

Post-mortem showed numerous cysts scattered over the brain. None observed in the skin. Eyes were not examined.

Pretoria General Hospital.

Dr. H. J. Hugo, Medical Superintendent, Pretoria Hospital, records a case of *Cysticercus cellulosae* in a European male, 33 years of age, who was admitted to the General Hospital suffering from concussion, due to a fall under epileptic seizure. The *Cysticerci* were diagnosed by X-Ray examination. This case was admitted on 31.14.36.

CONCLUSION.

Dr. Cawston of Durban is at least one authority who strongly suggests that writers on this particular subject should stress the importance that regulations should provide for the compulsory autopsy on every deceased epileptic in South Africa. The fact that Dr. Crosthwaite found in his limited observations *Cysticerci* were the exciting cause of 11·7 per cent. of epileptic cases, suggests that a fairly high incidence of human cysticercosis exists in South Africa. Dr. Crosthwaite's observations were confined to native cases, and, although the incidence of this fatal condition must be higher among natives, there can be a grave suspicion that numerous European epileptics may also be affected with cysticercosis.

All Europeans resident in countries in which the incidence of *Cysticercus cellulosae* is high in pigs, are in danger of contracting *C. cellulosae* through the *interim* adult *T. solium* stage in their own, or somebody else's person, by direct or indirect contact with that person. The known incidence of *C. cellulosae* in pigs and *T. solium* is said to be low in India, and in that country only the very lowest caste handle or touch pork, and yet relatively large numbers of Britishers serving in that country have contracted, what might be termed a pitiable disease, through contact with but a percentage of the Indian native population. Attention may be drawn, however, to notes which appeared in the *Indian Veterinary Journal* 3, p. 52 (1926-27), in which it was estimated that the incidence of *C. cellulosae* was 50 per cent. in Madras and Coimbatore in pigs.

One should not appear to be an unwarrantable alarmist, in comparing conditions in South Africa, with its much larger source of infection, namely a high percentage of porcine cysticercosis, a correspondingly suspected high incidence of *T. solium* among natives, and the fact that in approximately 100 per cent. of South African households the preparation and cooking of food is performed mainly by natives, a large percentage of whom may be presumed to be potential *T. solium* carriers.

PART VI.

The Eradication of Cysticercosis-Taeniasis.

A. The Necessity for Eradication.

Two factors which demand the eradication of cysticercosis are economic and hygienic. In a country such as South Africa it is essential that we should take the economic factor into serious consideration. The meat industry is becoming more and more important in this country, and we are trying to compete on the overseas markets with rivals, where the incidence of *C. bovis* is considerably lower than in South Africa, e.g., Australia, New Zealand, Canada, United States and the Argentine. In 1935, this factor was forcibly stressed by Irvine-Smith in the *Annual Report of the Director of the Abattoir and Livestock Markets, Johannesburg.*

"The Natal Agricultural Union has forwarded a resolution to the South African Agricultural Union recommending that the Government should introduce legislation to permit meat passed by Government Inspectors to enter Municipal areas without further inspection. All meat is, at present, inspected under the national standard of meat inspection laid down by the Minister for Public Health under the Public Health Act, by inspectors approved by the Government, and for the protection of Public Health is re-inspected on arrival in England, and is also further re-inspected on introduction into any local authority's area. In the event of measles being found overseas, in Union of South Africa meat on these re-inspections, which are essential, the South African export trade would receive a nasty jar. It is the responsibility of the farmer to eradicate measles." (Irvine-Smith, 1935.)

In the same report Colonel Irvine-Smith wrote further: "If you want to achieve success in the meat trade, you will have to eradicate measles. We had a recent example in Durban. Your competitors will exploit the question of measly beef. The opposition in the Argentine and overseas will at once say that you are feeding the housewife overseas with measly beef and they may get hold of some measly beef and ruin your trade. This is an aspect which should be seriously considered." Irvine-Smith also reminds his Council that chilling does not kill the *C. bovis*, therefore the presence of measles in export chilled beef is so much more undesirable.

It will be recalled that approximately 7,000 pigs are condemned annually in the South African abattoirs from which statistics were obtained. (See Incidence Survey, Part II.) Since measly pigs are usually totally condemned, this means a dead loss to the pig breeder or to the butcher of approximately £17,500 per annum, assuming the round average dressed weight of pigs slaughtered at our principal abattoirs to be 120 lb., and the average price per lb. dressed weight paid by butchers or auctioneers to be 5d., and applying these averages throughout the Union.

It is also estimated that nearly 7,000 bovines are annually found to be measly at Union abattoirs. On the assumption that all these bovines were to be condemned outright, and that the average dressed weight per bovine carcass was 600 lb., sold at an average price of of 25s. per 100 lb. for good medium beef, this would mean a loss of £52,500 per year to the beef industry. At this rate the total loss, that is through *C. cellulosae* and *C. bovis* would be £70,000 per annum.

This loss is, however, reduced by the freezing of approximately 80 per cent. of measly beef carcasses (lightly infested) at six of the principal abattoirs in the Union. Nevertheless, the average price paid for this frozen beef at those abattoirs is approximately 15s. per 100 lb., which represents a loss of about 10s. per 100 lb. on average good medium carcasses, not considering the cost of applying the freezing treatment imposed by some of our abattoirs. Despite this, the fact that a large percentage of our measly beef is not condemned outright, there is a considerable reduction in the value of such treated beef.

It is obvious that the figures showing the average cost of measles per annum to the meat industry, refer only to the losses incurred at approximately 65 Union abattoirs, from which incidence statistics were obtained. Financially, however, it is doubted whether the toll of measles is greater than the estimate of £70,000, since it can safely be presumed that no statistics were kept at those abattoirs not included in our lists in Part II, or else they would readily have been supplied by the authorities of many other towns, who were approached. There are also many smaller places in the Union, where cattle are slaughtered and consumed, but no inspection of any kind exists. Such cattle, therefore, whether measly or not are consumed by the unsuspecting public, and presumably top prices are paid—hence, no economic loss in small townships in the remote rural areas and in Native Territories.

The hygienic necessity for the eradication of cysticercosis is quite obvious, and has been fully discussed in the previous parts of this work. The fear of human infection is perhaps the most important and logical reason why in the larger centres, and in those smaller centres where proper meat inspection is carried out, efficiency in duty has at least been instrumental in breaking the life-cycle of those parasites which are found on meat inspection.

B. A Plan of Eradication of Cysticercosis-Taeniasis

1. *Co-operation, but not Encroachment.*

There should be closer co-operation in this important aspect between the members of the medical and veterinary professions, each of whom should be independently responsible for the destruction of the life-cycle at the respective stage which falls within his province.

This can best be elucidated by the old saying "Shoemaker stick to your last." In other words place the responsibility of destruction of the adult tapeworm upon the medical man, and that of the destruction of the bladderworm on the veterinarian. Close co-operation in this campaign need not necessarily lead to the encroachment by either profession on to the province of the other. At the present time, in South Africa in particular, there can be no gainsaying the fact that in many centres work which should purely be handled by veterinarians is being done by medical officers of health. I refer here to abattoir control, dairy control and control of inspections of meat emporiums. In South Africa there are at the present time only five municipalities which employ full-time veterinary officers, whose main duties are control of the respective abattoirs. In all other centres, including some of our bigger cities, the control of the abattoir is exercised by superintendents who have, or have not the certificate of the Royal Sanitary Institute in meat inspection, and these officials, who have no power to condemn meat, must call the medical officers of health, who in many cases know considerably less about diseased meat than the meat inspectors. In most of the larger centres where no veterinarians are employed, highly capable meat inspectors are employed, but most of these officers would preferably serve under the guidance of a veterinary officer, especially in intricate cases in which differential diagnosis involving cysticercosis is concerned. The urban meat consumer has the right to demand protection, and it should be made compulsory in all centres with a European population of 7,500, that a qualified town veterinary officer be appointed, who in smaller or larger centres could have control of the dairy inspection staff as well. It is not my intention to use this article as propaganda for the profession to which I have the honour to belong, but in many of the smaller urban areas (populations of 7,500) the main functions of the medical officers of health are the control of officials in charge of abattoirs and dairies, functions which could, with greater safety to the public, be performed by veterinarians. The position is not at all impracticable, and the fact that at present there are not sufficient veterinarians to take over such duties in all Union centres with populations of 7,500, does not mean that a number of young men will not take to the profession if sufficient inducement could be given. In the writer's opinion, as already stated, only co-operation between the medical and veterinary professions will eventually eradicate cysticercosis-taeniasis, hence the eradication of the bladderworm should be the function of the scientist best qualified for the purpose, viz., the veterinarian. Salaries of municipal veterinarians can be partly subsidised by Government, whose bounden duty it is to safeguard the health of the urban dweller. Thus, whilst eradication of the *Cysticercus* is the function of the veterinarian, that of *Taenia* must be done by the physician, who should be encouraged by legislation towards this important function, to obviate remarks such as those which Reitsma (1931) had occasion to use in Holland: "We must not lose sight of the fact that the object of the campaign against cysticercosis is the eradication of the *Taenia*. It is a remarkable fact that although veterinary scientists are paying a great deal of attention to cysticercosis, a state of lethargic rest exists in the medical camp as regards taeniasis, and one can obtain hardly any data regarding the disease either from the State public health authorities, or from private practitioners. The only facts the latter can state are that they have large and extensive practices, and once or twice a year they may treat a patient for tapeworm."

2. *More Thorough Meat Inspection.*

It has already been stated, in a previous Part of this article, that we in South Africa possibly permit a larger range of inspection incisions than is practised in most European countries. Nevertheless, even we can improve upon our technique. The present writer suggests the following technique in respect of examination for *C. bovis*:—

(*a*) Two long and parallel incisions into the masseters, on both sides of the face, in an upward direction, to completely sever the parotid gland below the ear.

(*b*) Two long incisions into the pterygoids, on each side.

(*c*) Numerous longitudinal incisions into the muscles of attachment of the tongue.

(*d*) Careful manual palpation of the whole of the heart; complete halving of the left ventricle; careful inspection of the myocardium.

(*e*) Careful manual examination of the oesophagus.

(*f*) A transverse incision into the hump, after the carcass has been cleft.

(*g*) Usual inspection of the viscera, without further incisions.

(*h*) A complete incision into the Triceps brachi and Deltoideus on each side.

(*i*) One incision into the Psoas muscles on each side.

(*j*) A deep incision into the Adductor muscle about an inch below and parallel to the symphysis pelvis.

(*k*) In the event of measles being found in any of the above locations, then the secondary incisions laid down by Public Health Act must be made.

It is doubtful whether the technique can be improved in respect of the inspection of pigs for *C. cellulosae*.

3. *Systematic Meat Inspection at all Abattoirs and Slaughter Poles.*

This suggestion is probably the most difficult to put into effect, and may even be considered impracticable. Reference will be made to the comments of the Town Clerk of Barberton, who mentioned that, whilst thorough inspection was being practised at Barberton, certain smaller townships in the vicinity were permitting the slaughter of bovines without any meat inspection, to the detriment of the Barberton stock-buyers. There is hardly a large or a small urban area in the Union, which is not faced with the same problems as Barberton.

I do not wish to emphasize in a dogmatic way that the Department of Public Health should appoint Meat Inspectors, who could be stationed at the urban centres of these small townships, and could from there do daily rounds of the slaughter poles of such small surrounding townships, but the matter certainly warrants the investigation of the Department. In many of these small townships daily slaughtering is not practised, and quite possibly such hypothetical inspectors could arrange for the slaughter days at the various small townships in their areas.

Such inspectors could either be paid by Government, or else the townships could be grouped and each group, subsidised by Government, could be responsible for the salary of its meat inspector. The Meat Inspectors must be qualified, holding the Royal Sanitary Institute's Meat and Other Foods Certificate. The aim should be the protection of all purchasers of meat, and the prohibition of unfair competition between dealers and butchers in the townships where no inspection exists, who slaughter measly meat without fear or scruple, and their less fortunate confrères in the more enlightened municipality close by, where up to date inspection is carried out.

An alternate suggestion may be to prohibit, *in toto* the sale of meat in any area controlled by a Village Management Board, Health Committee, or other form of Local Government, unless the meat had been slaughtered at a Public Abattoir, where proper meat inspection is practised. The small townships in the vicinity of an urban area would, therefore, be compelled to use meat slaughtered at a central abattoir (situated in the bigger town). The main idea at the back of such a scheme, it may be repeated, should be to safeguard, where possible, all purchasers of beef or pork from infection with taeniasis and to ensure that the unconscientious stock-raiser has no outlet for the sale of his measly stock, and thus to obviate the odious state of affairs mentioned by the Town Clerk of Barberton. (See Paragraph 4.)

Some Municipalities, e.g. Worcester, Mossel Bay, Burghersdorp, Clocolan, and to its utter disgrace, the fairly large City of Bloemfontein, permit the slaughter of pigs on farms, and the sale of the carcasses on the local market. The carcasses must be brought to the abattoir for inspection, with (in the case of Bloemfontein) the pluck attached—stomach, intestines and other viscera are not produced. In fairness to the Bloemfontein City Fathers, however, it must be mentioned that this arrangement was authorized by the Orange Free State Administration many years ago, and despite the efforts to have it rescinded by my colleague-predecessor and myself, it is still in force.

The reason why this was allowed is obvious—a Province existent on a purely farming industry naturally encourages that industry, although the more important aspect of Public Health is sadly overlooked. The results of this practice are clearly reflected in our observations at Bloemfontein. In two years we have found that 2·13 per cent. of pigs *slaughtered* at the abattoir have been measly, and yet, out of many hundreds of pigs slaughtered on the farms and brought to this abattoir for inspection, in three full years, my inspectors and I have found only two measly (both very lightly infected). Pigs, as we have seen, are more commonly very heavily infested, and obviously farmers do not bring to the abattoir for inspection, pigs which they see are measly, upon dressing. They have a ready sale for this measly meat to their natives, and a justifiable use for the lard for soap making. This statement is not made on mere conjecture, but is an actual admission of at least two farmers who occasionally patronise us with a few pigs for inspection. Here is an anomaly which can be immediately rectified by legislation. If the serious hygienic and economic importance of the disease were to be brought home to them, even the most ardent, and at times almost fanatical legislators, who vigilantly safeguard the interests of the farmer, will vote approvingly for the compulsory slaughter of all pigs intended for urban consumption, at urban abattoirs.

4. *The Prohibition of Insurance Schemes.—Loss must be carried by the farmer or producer.*

In a number of our larger Union abattoirs bovine cysticercosis is included in insurance schemes. Premiums are imposed on all animals to be slaughtered and the farmer or butcher is quite indifferent as to whether or not his ox is condemned. The direct result of these insurance funds is that the farmer does nothing to safeguard his cattle from infection, whereas it should be his compelled duty to realize his obligations. What the farmer does not realize, however, as Mönnig (1936) puts it, is that he after all, pays the insurance premium himself, and that abattoirs are not philanthropic institutions which willingly, out of sympathy for the unfortunate farmer, refund the price of the ox lost to him, without making him pay extra for the many uninfected oxen which have passed inspection.

It is questionable whether such insurance schemes serve any useful purpose, and at several of our larger Union abattoirs (Bloemfontein, Port Elizabeth and Capetown) they are totally discouraged. At others, e.g. Durban, where an insurance fund is conducted, the capitation fee is 3s. 6d. for cattle from Natal proper (in the case of Durban), but 5s. 6d. for cattle from so-called "black" areas, as Swaziland and the Natal native areas, where the infection is high.

In his Annual Report for the year ended 30th June 1935, the Director of Abattoirs, Livestock Markets, Veterinary Services, Ice and Cold Storage Departments, Johannesburg, refers to consignments of export cattle from Natal, received at Johannesburg, which showed infestation rates varying between 2·08 per cent. and 60 per cent. in thirteen consignments from different owners. Colonel Irvine-Smith adds: "The only manner in which the Council could assist in the eradication of measles in meat was to decline to indemnify cattle from proved sources of infestation. With this object in view, it was decided that after 1st July, 1935, any owner forwarding a consignment of cattle for export containing measle infestation to the Johannesburg Abattoir, would have measles or bladderworm infestation excluded from his indemnification until three subsequent consecutive consignments had been received from him free from infestation. The Director is of the opinion that when measle infestation is discovered in a consignment of cattle, the whole consignment from that particular owner should be debarred from export, otherwise subsidised measly beef from South Africa will eventually be found on arrival in England".

In considering this question from all points of view, the conclusion come to is that insurance schemes which include indemnification against measles, are definitely not in the interest of the country, and those of us whose calling assists towards the eradication of the menace of measles, should collectively press for legislation forbidding the inclusion of measles disease in abattoir insurance schemes.

As has been expressed, the farmer unwittingly pays an excessive slaughter fee (including his insurance capitation fee), which is quite needless, but he is perfectly satisfied as long as the payment of this capitation fee leads to his recovery of the price of any of his stock which may be condemned.

The practice at most abattoirs, where no insurance schemes are in vogue, is that loss through condemnation of carcasses for measles in bovines is borne entirely by the butchers, and not by the producers. In some cases butchers buy slaughter cattle from farmers out of hand, or at auction sales, and these cattle, if in good condition, may find themselves at the abattoir within a few days. In other cases butchers place nearly all their bovine purchases on stock farms, and they are withdrawn from time to time as the butchers' requirements dictate. Often, therefore, such slaughter bovines may run for several months on the butcher's own farm, among his reserve slaughter stock, in which case, after eventual slaughter and measles being found, the butcher will have difficulty in establishing scientific proof of the age and origin of infection. In those cases, however, in which bovines are slaughtered immediately, or within a few weeks of purchase, the butchers should have a " clear case ", in the event of measles infestation being found. Measles disease is, and should in every case be considered by buyers themselves, a latent defect, if found in stock slaughtered within a reasonable time after purchase. In this respect butchers can assist towards the eradication of measles, if they would all decline to pay farmers for such infected purchases. Unfortunately competitive buyers have spoiled the producers, with the result that at some places the butchers who insist on a measles-free guarantee from farmers are frequently ousted by their more generous competitors. This fact is very much in evidence in Bloemfontein, with the result that nearly all local butchers will suffer the loss of a purchase through measles, rather than provoke the displeasure of their sellers, who would immediately supply their rivals with stock, to their exclusion, should a refusal to pay for condemned measly cattle have occurred.

A similar state of affairs is related by the Town Clerk of Barberton, whose most interesting memorandum, dated 27.10.36, and the very useful suggestions it contains may be mentioned almost *verbatim*:—

" It is fair to state that in rural centres, such as Sabie, Noordkaap, Sheba, Eureka, Louwscreek, Hectorspruit, Komatipoort, Kaapsche-Hoop and Nelshoogte, animals are slaughtered in abattoirs where no post-mortem examinations are made. In these rural centres where slaughtering is carried on, not under the exigencies of meat inspection, the percentage of bovines infested with this parasite must be just as high as is found in the Barberton Municipal Area (i.e. about 5·31 per cent.), as only a negligible amount of stock is local, the greater proportion being bought in the districts where this slaughtering takes place. This being the case it seems that condemning a carcass infested with measles in the Municipal Abattoirs is a needless procedure in eradicating either measles in cattle or tapeworm in man. It certainly protects the urban residents from contracting the parasite and as such renders these people safe, but still the incidence in cattle is on the increase; this is due to the lack of systematic uniform inspection of meat at *all* slaughter poles. On several occasions this situation has been discussed by the butchers and our Health Committee, and it appears that if the butchers wish to obtain recompense for losses suffered from the farmer or stock owner, in practically every instance he is told ' if you do not wish to buy my stock without making me responsible for your losses I shall simply sell to other buyers in areas where no inspections are carried out. They never suffer losses '.

In one instance of ' X ', a most progressive attitude was adopted. He made himself responsible for half the loss the buyer of his stock had suffered, and immediately had all the natives on his estate examined for evidence of being the unwilling host of the tapeworm. Within a short time twenty were found to be harbouring this parasite. Thereupon he made a strict rule that all infested natives employed by him had to be successfully treated to remove the entire tapeworm, and furthermore that every new native hired by him had to submit to an examination. Any native breaking this rule would be dismissed from his service. This appears to be a very good measure for dealing with the problem and were all ranch owners, cattle breeders and farmers equally progressive and willing to help in the eradication of the parasite, the whole position would be materially improved.

As it is, legislation to enforce this result would have to be passed in such a manner that a high infestation of measles in the stock of any particular owner would reflect materially on that particular individual. If an owner were careless about sanitary measures, careless whether his cattle became infested or not, and did not feel the loss in any way, when on post-mortem examination they were found to be infested, and legislation were passed that he stands the loss when a bovine is condemned, much would have been done to ameliorate the position. It is suggested that measures be taken to improve sanitary conditions on his farm, free the hosts of their parasites and examine every new employee. The farmer should be brought to realize his responsibility in the matter and *all insurance schemes should be abolished*. In Barberton, as pointed out, the farmer, by reason of his many markets refuses to recognize his responsibility and the butcher, who must have stock, has to bear the loss himself, and only a few miles away his competitors buy and slaughter without thought as to infestation and with no fear as to monetary loss.''

These remarks by the Town Clerk of Barberton embody several of the suggestions made in previous paragraphs, but, in order to use his memorandum in a concise form, without referring to extracts here and there, the memorandum has purposely, as it is, been embodied at this stage of our discussion.

5. *Avoidance of all possible sources of contact of the susceptible animals with human dejecta.*

Pigs should be kept in sites, and on no account must they be permitted to roam about the farmyard.

Suitable latrines or privies should be constructed on all farms for the use of Europeans, and separate latrines for natives. The latter may be constructed close to natives' quarters. Meat inspection, alone, will never successfully eradicate taeniasis, if we do not safeguard infection of our meat animals with *Taenia* eggs. Our primitive Europeans and our native population must be educated in the first principles of hygiene. This campaign may be difficult, but the obstacles are not insurmountable.

What would appear to be quite a practical suggestion, is the fencing off of strips of veld, within which natives may defaecate. Such narrow fenced strips, with narrow inlets, so that cattle cannot enter them, can be provided on various parts of the farm, especially close to such parts where the farm labour is most frequently required, and also in grazing camps for the herd-boys, etc. Such fenced surface latrines may not, however, overcome the possible spread of *Taenia* ova by such agencies as water, insects (dungbeetles, blowflies, etc.), birds, etc. The provision, therefore, of trench latrines, and the enforcement of the immediate covering up of the deposited excretum by the native may thus be more effective, although it may be a trifle more costly in money and labour. Covered bucket latrines are, undoubtedly the most effective.

On no account should slaughter bovines be grazed on lands fertilized by human excretum, sewage farms, etc., and the use of fodder (e.g. lucerne, etc.) grown on such lands must be entirely discouraged.

The Native Affairs Department, through its Extension Officers, can by lectures to tribes, assist towards the eradication of the parasite.

It is astonishing how little the general public knows of the life-history of the parasite. Our campaign must, therefore, be directed at the rural source of the disease. All three Departments interested, namely Agriculture, Native Affairs and Public Health, can collectively assist in the eradication of the parasite. The Department of Agriculture can take more serious notice of the scourge by encouraging or instructing its Veterinary Officers, Stock Inspectors and Extension Officers to lecture groups of farmers on elementary farm hygiene. If Extension Officers and Stock Inspectors have no scientific knowledge of Cysticercosis-taeniasis, this can soon be taught to them, by brief courses at Onderstepoort, or by arrangement, at most of the principal abattoirs where qualified veterinary surgeons are employed. Armed thus with Departmental Pamphlet-Bulletins, the officers of the Department of Agriculture can disseminate the necessary knowledge to groups of farmers' meetings, where these bulletins in both official languages can be distributed. Our farmers will then receive sufficient enlightenment to attack the parasite by means of the most primary weapon, that of farm hygiene, hygienic prophylaxis and the means of maintaining this prophylaxis.

Similarly Extension Officers of the Department of Native Affairs and Sanitary Inspectors, seconded for service in native areas, may deliver lectures in Native Reserves. Enlightenment of all concerned is what is most urgently required, and the writer has, in the course of his former Government veterinary duties, often experienced that Native Chiefs are ever ready to co-operate in campaigns concerning the health of their live stock—their only real token of wealth or possessions. Good work, through enlightenment, has been accomplished by co-operation of native tribes in other veterinary campaigns in this country, e.g., the campaigns against East Coast fever, Scab and Anthrax, and if we did not have the whole-hearted co-operation of our native co-dwellers on our borders, we would not have effectively stamped out the few outbreaks of Foot and Mouth Disease which occurred, or prevented its further entry into the Union, in a remarkably short space of time, during 1933. This may all sound idealistic, but many of our present-day Native Chiefs are quite intelligent, and if instructed to do so, they may be trusted to enforce strict hygienic sanitary laws among their people. The trouble is that they have never yet, as a plan of campaign, been *requested* to do so, nor has the *necessity* for the enforcement of tribal latrine arrangements been brought home to them. The present writer considers that this is an experiment well worthy of a trial.

6. *Free medical treatment of* Taenia *carriers.—Rewards for Production of* Taeniae *by such carriers.*

The enlightenment of our farming and native populations should next be followed by free medical treatment. Liquid extract of male fern, or whatever vermifuge the Public Health Department may recommend should be available for all *Taenia* carriers, whether on farms, native reserves, or in towns. Magistrates, District Surgeries or Dispensaries, Justices of the Peace in rural areas, or Native Chiefs should be provided with quantities of the drugs required, and careful directions for use may be given to those who have to dispense the drugs and may have no knowledge of therapeutics. If the campaign were to end with the free treatment of *Taenia* subjects, these must be told that evacuated *Taeniae*, segments, etc., must be burned or buried, but must not be discarded where live stock can come in contact with the dejecta.

Instead of ending the campaign with the free treatment of known carriers, it might be more advisable to encourage, in some form or other, this free treatment. In Australia and in Germany (in Wurtemburg, and lately throughout the country) rewards have been offered for the production of every tapeworm or piece with the head. In this connection, Dr. Heinrich Wagemann of Flensburg, writes (letter dated 22nd November, 1936): " For every tapeworm or piece with the head, which is sent to the physician of the Government Health Department, the tapeworm carrier is paid 10 R.M. Only by these measures, I think, we shall be able to fight measles, and we hope that these measures, even after a considerable period of campaigning, will ultimately eradicate measles in cattle altogether ".

It is possible that such a campaign may cost a considerable amount of money, if applied in South Africa, but, in consideration of public health and the loss to the meat industry locally, as well as to the menace to our potential chilled beef overseas market, it may well warrant the offer of small rewards to all those, European or native, who produce either the complete *Taenia* or portions including the head. Perhaps free treatment, plus 2s. 6d. for each head may be a big incentive to our poorer Europeans and natives to rid themselves of their health-destroying guests. Rewards have been offered by the Government for the production of evidence of destruction of other vermin, e.g., the brushes of jackals, and it is quite possible that the economic loss from these marauders has been less than that caused by the tapeworm carrier.

It may be interesting to relate that Hall (1927) wrote: " As one by-product of that work, it might be mentioned that following the hookworm campaign of treatment and sanitation in Panama, the incidence of *C. cellulosae* in swine at the Panama City abattoir dropped from 15 per cent. to 5 per cent., according to the inspector, Dr. Mattatall. This 10 per cent. reduction in condemnations resulted in an annual saving of 40,000,000 dollars, a very valuable by-product of a hookworm campaign."

A campaign directed against the tapeworm in South Africa, and not necessarily the by-product of some other helminthic campaign, may, in a few years, show a remarkable economic result.

In Switzerland in 1917, and in Germany quite recently, attempts have been made to trace a *Taenia* carrier through infection found in a bovine carcass at the abattoir. The origin of the infected bovine is traced. In this connection Dr. Wagemann (Flensburg) writes (22.11.36): " Since August, in Germany, we also search for the tapeworm carrier. Thus, when measles disease is found, the Police trace back the origin of the animal. It is sometimes found that the owner of the animal, or a member of his household is a tapeworm carrier. In case such a person is found, he is compelled to have himself treated by a physician." In South Africa the tracing of a tapeworm carrier, in this way, may be impossible, owing to our large areas and the fact that frequently the origin of a bovine cannot be traced, since before slaughter it might have changed hands several times. The tracing of the origin of a *Taenia solium* carrier may be more practicable, except in measly pigs forwarded by speculators, since pigs delivered at abattoirs are frequently reared by the consignors.

In conclusion, it is of value to insert here a translation of Forms " A " and " B ", at present used in Germany in connection with that country's campaign against Cysticercosis-taeniasis.

Direction of Abattoir, or
Veterinary Meat Inspector

FORM A.

(name)

Place.............................. Date............

To the Sanitary Police in..

for transmission to..

(name and address of owner of animal)

Re: Information regarding Measles in Cattle.

From the consignment of........................, in......................,

(bovine or calf)

from the butcher................, in................, dated...........193...

(address)

which was slaughtered and was diagnosed to be measly on examination.

The beef measle is the intermediate stage of the development of a human tapeworm. The finding of measles in an ox appears to prove that a tapeworm must exist in a human being in the particular part from which the animal comes from. It is essential that investigation should be made whether the owner or one of his family, or somebody in his employ is infected with a tapeworm. The symptoms are: constipation, gastric derangement, loss of appetite, temporary listlessness, constipation followed by diarrhoea, and the finding of segments of tapeworm in the excretum. Accordingly, you are requested to see that the tapeworm carrier submits him/herself to medical treatment. For every tapeworm, or part with *HEAD* attached, found as the result of medical treatment, and forwarded to the Health Department, Veterinary Division, 82/84 Unter den Eichen, Berlin, preserved in spirits, with the attached form B completed, the tapeworm carrier will be paid 10 R.M. (ten Reichsmarks) reward by the Government.

....

(Signature of the Abattoir Director/or
Veterinary Meat Inspector.)

FORM B.

To be filled in by the Physician.

Place.............................. Date..............

District..................................

INFORMATION REGARDING FINDING OF A TAPEWORM.

According to the attached information from........................
the Abattoir Direction
Veterinary Meat Inspector

......................at......................dated...................., I have found that this person...,
(owner of animal)

of...................... ....found a tapeworm, and
(full address)

I am sending this in spirits, with head attached.

....

Examining Physician.

To the Government Health Department,
Veterinary Division,
82/84 Unter den Eichen,
Berlin-Dahlem.

EPILOGUE.

1. From records obtained from the large majority of abattoirs in South Africa, it was gleaned that the incidence of *C. cellulosae* in pigs varies between a fraction of a percentage and 25 per cent. These percentages represent averages varying from one year's to ten years' observations.

From only three Union abattoirs were percentages of less than 1 obtained. Average percentages of 5, or over, were obtained from no fewer than 24 Union abattoirs, and of this number 7 returned percentages in excess of 10. A definite " black " zone is traceable on the map of the Union, extending from Vryburg and Mafeking in the North-West to Nelspruit in the North-East, passing through the whole of the central Transvaal, via Lichtenburg. Potchefstroom, Rustenburg, Pretoria, Witbank and Middelburg. A similar " black " zone is traceable along the whole of the eastern border of the Orange Free State (bordering on Basutoland), and this includes the areas which supply the abattoirs at Wepener, Clocolan, Ficksburg, Senekal and Bethlehem. In the Cape Eastern area heavy infection was also found at three abattoirs, Fort Beaufort, Kingwilliamstown and East London, which probably draw pigs from the Transkeian Territories.

The incidence of *C. bovis* is much lower than that of *C. cellulosae* in South Africa. From 18 abattoirs were percentages of less than 1 obtained. From only 9 abattoirs were average percentages of 5 or more obtained. The highest percentage infections were obtained from the far Eastern Transvaal (bordering on Swaziland), that is Barberton, from those Natal abattoirs which draw a large amount of slaughter bovines from Zululand, and from the four Cape Eastern abattoirs, East London, Kingwilliamstown, Fort Beaufort and Port Elizabeth.

In general it would appear that there is a slight decrease in the incidence of *C. cellulosae* in the Union of South Africa, but a decided increase in that of *C. bovis*, during the last few years.

2. *C. cellulosae* infestation in South Africa usually assumes a very heavy, generalized nature. Approximately 5:1 may be taken as a fairly indicative ratio of heavy infestation to light infestation.

In the case of *C. bovis* infestation, the reverse is the case.

3. On account of the usual heavy infestation in pigs, it is not customary to describe definite predilection sites, but in bovine infestation it must be stressed that the muscles of the hind quarters, in addition to the common " predilection " sites found by workers in Europe, are very frequent locations of infection. The hind limbs are not incised in measles inspection, and it is recommended that attention should be drawn to this important predilection site. Incisions can be made deeply into the adductor muscles, parallel to and just below the pelvic symphysis, without mutilating the quarter. The hump is another important site of infection, and regulations should provide for the incising of this area.

4. Thorough inspection technique is seriously advocated. At Bloemfontein it was found that thorough inspection, coupled with long and deep incisions into the prescribed sites, was rewarded by the finding of four times as many measly carcasses, compared with the figures recorded prior to the adoption of our technique, that is before my Senior Inspector and I assumed office, simultaneously, at this abattoir.

5. In South Africa, it would appear that the origin of infection in the pig is the same as in almost all other countries, in which a fairly high incidence of *C. cellulosae* still occurs. On primitive farms and in native locations it is customary to allow pigs to wander about the farmyard, or in the vicinity of human habitation. On many such farms and Native Reserves the most primitive hygienic arrangements exist. The pig, under such conditions, readily acts as a scavenger and becomes heavily infested.

It has been observed that the incidence of *C. bovis* is higher during, or just after, periods of drought, when there is little grazing on the veld, and when, consequently, bovines tend to remain near human habitations. The incidence of infection is least among cattle drawn from vast ranges. Streams play a small part in the dissemination of *T. saginata* ova in South Africa. Stagnant pools in rivers used as watering places for cattle, may be suspected as potential points of danger. Sewage contamination of grazing lands has not been observed as a serious source of infection in this country, and actual records such as those from Germany, Holland and Australia are not available in South Africa.

6. Actual viability tests were performed during twelve months by myself and my staff at the Bloemfontein abattoir, and the results obtained varied very slightly from those obtained by workers in Europe, but differed greatly from the extremely negative results obtained by Annie Porter, who was the only South African worker who had previously attempted such viability tests.

The results of our tests showed that *C. cellulosae* can survive the death of its host, and evaginate its scolex (tested by Keller's method) by at least 41 days, under ordinary chilling room conditions. *C. cellulosae* may, in exceptional cases survive four days' continuous freezing at approximately -10° C., in a shoulder of pork weighing 15 lb.

Similarly, in an exceptional case it was proved that *C. bovis*, subjected to 5 days' continuous freezing in a side of beef weighing 288 lb. was still capable of evaginating its scolex by Keller's method.

In no case was it found that *C. cellulosae* survived a period in excess of four days' freezing, or *C. bovis* in excess of five days' freezing.

7. Judging from personal information obtained from various medical observers, there is sufficient evidence to presume that both species of tapeworm are relatively common among our native population, and not at all rare in Europeans in South Africa, especially in rural areas.

Furthermore, Dr. van Coller, working in South Africa found that a large percentage of cases of psychosis were due to tapeworm infection.

Comparatively large numbers of cases of epilepsy in humans have been found, on post-mortem examination to have been due to *C. cellulosae* in the brain, etc. Dr. Cawston of Durban asks that compulsory autopsies should be held on all deceased epileptics.

CYSTICERCOSIS IN SWINE AND BOVINES.

8. Cysticercosis in swine and bovines is a costly scourge to the agricultural industry of South Africa, and taeniasis is a serious and disgusting infection in its population. Together, the two diseases caused by two stages of a common parasite should receive the full and collective attention of the veterinary and medical professions. Ultimate eradication is *not* impossible although many years of costly campaigning may be entailed. The economic and hygienic results expected from such a campaign warrant the assistance of the State.

Cysticercosis-taeniasis can be eradicated by:—

(*a*) Close co-operation between the veterinary and medical professions.

(*b*) Closer inspection of swine and bovine carcasses at abattoirs in the larger urban areas, with, possibly, veterinary control in towns with more than 7,500 Europeans.

(*c*) Compulsory meat inspection at all other slaughter poles, by qualified meat inspectors. These slaughter poles to be grouped and the inspector stationed in the central urban area must control its inspections. Butchers in small villages, where no inspection exists must not unfairly compete with their colleagues in the larger urban areas, who suffer losses from condemnation. The farmer must have absolutely no loophole for the sale of his measly stock, without suffering the loss.

(*d*) Education of Europeans and natives in elementary hygiene, embodying studies of the life-histories of the two parasites. The assistance of extension officers, stock inspectors and sanitary inspectors can be obtained, to further this elementary teaching.

(*e*) The abolition of all insurance schemes, which include indemnification for measles, at abattoirs. These insurance schemes, which serve no useful purpose, have the effect that the careless farmer shows no appreciation of his responsibility for safeguarding his stock from infection.

(*f*) The compulsory slaughter of all pig carcasses intended for sale on urban markets, at urban abattoirs. The custom of many unconscientious farmers who dispose of pig carcasses they have noticed on dressing to be measly, to their unfortunate natives, is very strongly deprecated.

(*g*) Free treatment of *Taenia* carriers, and the offer of rewards to all such treated carriers for the production of either an entire tapeworm, or a portion with its head attached.

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Section III.

Plant Studies
AND
Poisonous Plants.

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Recent Investigations into the Toxicity of known and unknown Poisonous Plants in the Union of South Africa. VIII.

By DOUW G. STEYN, Section of Pharmacology and Toxicology, Onderstepoort.

CHENOPODIACEAE.

Atriplex semibaccata R.Br. O.P.H. No. 7030; 30.9.36).

Common name: Creeping salt bush.

Origin: Smithfield.

State and stage of development: Wilted and in seeding stage.

The plant was suspected of having poisoned sheep.

Picrate paper test for hydrocyanic acid.

A. *Wilted plant.* (10 gm. used in each test).

(*a*) Leaves: Strongly positive after 12 hours, discolouration of the picrate paper setting in within half an hour after insertion.

(b) Leaves + chloroform: Negative after 24 hours.

(*c*) Leaves + emulsin: Same result as in (*a*)—positive.

B. *Plant leaves and stems dried in shade and again tested for the presence of hydrocyanic acid.*

(*a*) Leaves and stems (5 gm.): Negative after 24 hours.

(*b*) Leaves and stems plus chloroform: Negative after 24 hours.

(*c*) Leaves and stems plus emulsin: Strongly positive after 24 hours, discolouration of the picrate paper setting in within half an hour after insertion.

The chloroform and process of air-drying apparently inactivated the enzyme responsible for the liberation of hydrocyanic acid from the cyanogenetic glucoside.

The air-dried plant (stems and leaves) was drenched to a sheep as follows: —

Sheep 45360 (*4-tooth*, 45 *Kg.*): 600 gm. in two daily doses on each of the 7.10.36, 8.10.36, and 9.10.36 and 300 gm. on the 10.10.36. Total amount of plant drenched – 2,100 gm.

Result: Negative.

As the plant submitted for investigation was found to contain a small amount of hydrocyanic acid we should certainly consider it possible that it may under certain soil and climatic conditions develop dangerous quantities of this poison.

Subsequent to the above experiments a small quantity of seed of the plant was obtained from Dr. M. Henrici, Veld Reserve, Fauresmith, and sown in the poisonous plant garden at Onderstepoort. On the 9th December, 1936, when the plants were about 2-4 in. high and flowering, material (whole plant) was collected at 12 noon on a hot and dry day for purposes of testing for the presence of hydrocyanic acid. There was a slight brownish discolouration of the picrate paper eighteen hours after insertion into the tube. The plants showed no signs of wilting. Unfortunately the amount of plant material available was insufficient to allow of tests to which chloroform and emulsin could be added. The plants are, however, showing good growth and these tests will be conducted from time to time.

CRASSULACEAE.

Crassula expansa Ait. (O.P.H. No. 6449; 21.9.36).

Common name: ———

Origin: Wolwefontein, C.P.

State and stage of development: Fresh and in the flowering stage.

Rabbit A (2·0 *Kg.*): 50 gm. of the fresh plant per stomach tube on 21.9.36; 50 gm. of the fresh plant per stomach tube on 22.9.26.

Rabbit B (2·0 *Kg.*): 80 gm. of the fresh plant on 21.9.36; 80 gm. of the fresh plant on 2.9.36.

Sheep 45360 (*Full-mouth*, 42 *Kg.*): 800 gm. of the fresh plant per stomach tube on each of two consecutive days.

Result: Negative.

EUPHORBIACEAE.

Acalypha indica L. (O.P.H. No. 2730; 7.7.36).

Common names: Indian Acalypha; Mukta-jhuri (Indian). Native name (N. Transvaal)—Machelikoane.

Origin: Vetfontein, Northern Transvaal.

State and stage of development: Slightly wilted and in the flowering stage.

Uses: According to the British Pharmaceutical Codex (Editorial, 1934) the plant is a gastro-intestinal irritant. In small doses it acts reflexly as an expectorant and in large doses as an emetic. It has been used as a substitute for Ipecacuanha. The natives in the Northern Transvaal use the plant in the treatment of eye-diseases.

Constituents: It contains an alkaloid, acalyphine, and resin, tannin and volatile oil. (Editorial, 1934, and Wehmer, 1929.) Mr. G. Roets, B.Sc., of Onderstepoort found that the plant contains hydrocyanic acid. An article on the cyanogenetic glucoside present in the plant is being prepared by him and Dr. C. Rimington for publication in this Journal.

Rabbit A (2·0 *Kg.*)*:* 10 gm. of the slightly wilted plant per stomach tube at 11 a.m. on the 7.7.36. There was laboured respiration within two hours after drenching.

8.7.36: Apathetic, not feeding, laboured respiration, and accelerated but forceful heart-action.

9.7.36: Died at 7 a.m.

Post-mortem appearances: Blood of a dirty chocolate-brown colour and not coagulated; pronounced oedema of the lungs; pronounced fatty degeneration of the liver; gastric mucosa covered with a thick layer of mucus. Stomach contents positive for hydrocyanic acid.

Rabbit B (2·5 *Kg.*)*:* 20 gm. of the slightly wilted plant per stomach tube at 11 a.m. on 7.7.36.

There were pronounced dyspnoea and restlessness within one hour after drenching. The heart-action was very much accelerated and became progressively weaker. Paresis and paralysis also set in commencing in the front-quarters and progressed until the animal was prostrate and unable to rise. Dyspnoea was very pronounced. The animal struggled continuously and died with convulsions, probably due to asphyxia, one and a half hours after drenching.

Post-mortem appearances: Blood of an intense chocolate-brown colour and not coagulated; all internal organs of a light dirty-brown colour; pronounced hyperaemia and oedema of the lungs; very pronounced hyperaemia of the gastric mucosa; the mucosa of the stomach and small intestine covered with a large amount of mucus.

Rabbit C (2·1 *Kg.*): 5·0 gm. of the dry plant (dried in shade) per stomach tube daily from 12.8.36 to 27.8.36.

Excepting loss of appetite, no symptoms of poisoning were seen in the animal until one hour after the 5-gram dose administered on the 27.8.36. There were pronounced restlessness, dyspnoea, and general convulsions with the head drawn backwards. Death occurred in a state of paralysis one and a half hours after the last dose on 27.8.36.

Post-mortem appearances: Pronounced oedema and slight hyperaemia of the lungs; heart in systole; slight sub-acute catarrhal gastro-enteritis; urine of a reddish colour; blood dark brown in colour and not coagulated. Stomach contents positive for hydrocyanic acid.

Rabbit D (2·45 *Kg.*): 7·5 gm. of the dry plant per stomach tube at 9 a.m. on 12.8.36.

The animal developed symptoms of poisoning similar to those described in rabbits A and B and died fifteen hours after drenching. The post-mortem appearances were also similar. The urine was of an intense reddish-brown colour and fluorescent.

Rabbit E (2·75 *Kg.*): 12·5 gm. of the dry plant per stomach tube at 9 a.m. on 12.8.36.

The animal died one and three-quarters of an hour after drenching. The symptoms and post-mortem appearances were similar to those described above. There was a number of haemorrhagic patches on the gastric mucosa.

Rabbit F (2·1 *Kg.*): 12·5 gm. of the dry plant per stomach tube on 12.8.36.

Death occurred one and a half hours after drenching with symptoms similar to those described above.

Post-mortem appearances: As described above and in addition extensive haemorrhage into the duodenal mucosa, which showed pronounced swelling; pronounced hyperaemia of the gastric mucosa.

The dark chocolate-brown heart blood was collected and examined spectroscopically by Dr. J. I. Quin, Professor of Physiology, Onderstepoort. There was no haemolysis and no methaemoglobin bands were detectable. It should, however, be pointed out that owing to the intense dark-brown discolouration of the blood it had to be diluted excessively in order to examine it spectroscopically. It is, therefore, possible that the dark-brown discolouration of the blood was due to the formation of methaemoglobin in spite of the fact that the spectroscopic analysis was negative.

As hydrocyanic acid is not known to cause such an intense dark chocolate-brown discolouration of the blood it was thought that the plant may contain a second, or even a third, active principle causing the discolouration of the blood and the gastro-intestinal irritation. There seems little doubt that the most pronounced dyspnoea seen in the experimental animals is due not only to the toxic effects of hydrocyanic acid but also to the discolouration of the blood, which most probably reduces its oxygen-carrying capacity.

An experiment was therefore planned in order to determine whether the plant freed from hydrocyanic acid was still toxic to rabbits.

The dry plant was ground, mixed with emulsin and moistened. It was then incubated at ±40° C. for a few days until only the slightest trace of hydrocyanic was left and again drenched to two rabbits as follows.

Rabbit G (2·5 *Kg.*): 15 gm. of the dried incubated plant on each of four consecutive days.

At no time were any symptoms of poisoning discernible.

Rabbit H (1·9 *Kg.*): 10 gm. of the dried incubated plant on each of four consecutive days.

The animal developed no symptoms of poisoning.

From these results it appears that the active ingredient, or ingredients, responsible for the production of the dark chocolate-brown discolouration of the blood and the gastro-intestinal irritation has also been inactivated by the method applied to expel the hydrocyanic acid from the plant.

GRAMINEAE.

Cynodon Transvaalensis Burtt-Davy.

Common name: Transvaal kweekgras; quick grass.

Origin: Warmbad, Transvaal.

State and stage of development: Fresh and flowering.

In spite of the fact that the plants showed no signs of wilting they were found to contain a fair amount of hydrocyanic acid.

Melica decumbens Thunb. (O.P.H. No. 14679; 5.3.37).

Common names: Dronkgras, Kaapse dronkgras.

Origin: Tierhoek, Klipfontein Siding, Cape Province.

State and stage of development: Dry and in the seeding stage.

The owner of the farm, Tierhoek, alleges that the above grass is the cause of " dronksiekte " (" intoxication ") in his stock. The symptoms described by the owner resemble those seen in cases of poisoning with *Cynanchum africanum*, *Cynanchum obtusifolium* and *Equisetum ramosissimum*, but the owner is convinced that none of these plants are to be found on his farm.

Each of sheep 41929 (6-tooth, 45 Kg.) and 46981 (6-tooth, 47 Kg.) received 4·4 Kg. of the dry grass per stomach tube over a period of twelve days without suffering any ill-effects whatsoever. The animals were chased about at intervals as exertion is known to precipitate the symptoms.

They refused to take the grass voluntarily.

Seed has been collected and will be sown in spring. It is intended to graze animals on the green grass.

Sorghum vulgare Pers.

Common names: Kafferkoring, kaffir corn, broom corn, shallu, durra, sorghum.

Origin: Vryburg, Cape Province.

State and stage of development: Dry immature and mature seed heads.

Hydrocyanic acid test (picrate paper).

(*a*) *Stalks.*

(1) Stalks (10 gm.): Negative after 24 hours.

(2) Stalks (10 gm.) plus emulsin: Positive after 24 hours.

(*b*) *Seed (mature and immature).*

(1) Seed (10 gm.): Negative after 24 hours.

(2) Seed (10 gm.) plus emulsin: Negative after 24 hours.

LEGUMINOSAE.

Canavalia ensiformis D.C. (O.P.H .No. 7007; 9.10.35).

Common names: Swaardboontjies; sword bean.

Origin: Mr. B. van der Vyver, Government Veterinary Officer, Pretoria, submitted a small quantity of the mature beans and wished to know whether the plant could be utilised *ad lib.* as a stock feed.

Ox 7063 (1 *year old;* 177 *Kg.*) *and Ox* 7133 (1 *year old;* 162 *Kg.*) These animals were fed with the entire plant in the fresh state. Feeding was commenced when the plant was in the early flowering stage and continued until the pods were almost mature.

The animals ingested 275 Kg. and 310 Kg. of the fresh plant respectively in a period of forty days. At no time was there any evidence that the plant exerted detrimental effects on the health of the animals. On the contrary, in spite of the fact that they took no additional feed they grew well and gained in weight.

At the commencement of the experiment ox 7063 did not take the plant too well, whilst ox 7133 relished it right from the first day.

It should be mentioned that some years ago the author fed the mature beans to a young bovine. The animal ingested 22 Kg. in a period of three weeks without suffering any ill-effects.

The following figures are quoted from Wehmer (1929):—

(*a*) *Seed:* 8-13 per cent. water; 22·7-26·8 per cent. protein; 2·6-3 per cent. fat; 1·1-6·6 per cent. cellulose; and 2·25-3·83 per cent. ash.

(*b*) *Pods:* 35·7 per cent, protein; 10·2 per cent. cellulose; 2·45 per cent. fat; and 3·33 per cent. ash.

Medicago sativa L.

Common names: Lusern; lucerne; alfalfa.

Origin: Plant material for the tests was collected from a small patch of lucerne growing on red sandy soil in a private garden (House No. 36) at Onderstepoort. For a period of eight days prior to the date on which the under-mentioned tests were made the weather was very hot and dry with the result that the lucerne, which was in the early flowering stage, showed signs of wilting.

Hydrocyanic acid test (picrate paper).

Specimens of the wilted lucerne picked at 10 a.m. on 8.11.36 and tested immediately showed the presence of fair amounts of hydrocyanic acid. Tests conducted with specimens of fresh lucerne collected from the same plot a few days after heavy rains had fallen were, however, negative.

It, therefore, appears that lucerne, like so many other plants (Gramineae, etc.) is likely to develop hydrocyanic acid during the process of wilting. Further experiments are in progress at Onderstepoort in order to go more fully into this phenomenon. The various Agricultural Colleges in the Union of South Africa have kindly agreed to co-operate in this matter and it is hoped that with their assistance and collaboration we will be able to collect valuable information. It may prove that death caused by lucerne is not always due to hoven as such, but that, in some cases at least, hydrocyanic acid may be a contributory factor. It is well known that hoven is a typical symptom of poisoning with this acid.

Tephrosia macropoda E. May. (O.P.H. No. 6139; 11.9.36).

Common Names: Zulu—iHlozana, iLozana, u Qwengu. Used as a fish poison by Natives.

Origin: Kelso Junction, Natal.

State and stage of development: Wilted and in the flowering stage.

The plant is suspected of having caused death in sheep.

Hydrocyanic acid test (picrate paper).

5·0 gm. of the wilted leaves plus emulsin caused dark reddish-brown discolouration of the picrate paper within six hours.

It is, therefore, possible that the plant may under certain conditions cause hydrocyanic acid poisoning.

Tephrosia Vogelii Hook.

Common names: Fish-bean.

Origin: Onderstepoort Poisonous Plant Garden.

State and stage of development: Fresh and in the pre-flowering stage.

Hydrocyanic acid tests (picrate paper).

(1) Fresh plant: Negative after 24 hours.

(2) Fresh plant + chloroform: Negative after 24 hours.

(3) Fresh plant + emulsin: Negative after 24 hours.

Tests will be made with the plant in the wilted state.

LILIACEAE.

Ornithogalum Pretoriense Bkr. (O.P.H. No. 10198A; 7.12.36).

Common names: ————.

Origin: Meyerton, Transvaal.

State and stage of development: Fresh bulbs and leaves in the post-flowering stage.

Rabbit A (1·9 *Kg.*): 60 gm. of the fresh bulbs and leaves per stomach tube on each of two consecutive days.

Rabbit B (2·15 *Kg.*): 120 gm. of the fresh bulbs and leaves per stomach tube on each of two consecutive days.

Result: Negative.

This plant has in the past been repeatedly drenched to sheep and rabbits with negative results.

Urginea sp. (O.P.H. No. 5603; 23.9.36).

Common names: ————.

Origin: Sekukuniland, Transvaal. Per Mr. A. O. D. Mogg, Botanist, Division of Plant Industry.

State and stage of development: Fresh bulbs with no leaves, flowers or seed.

The fresh bulbs were finely minced and then forced through a press with fair-sized holes (meshes). The expressed juice was dosed to rabbits.

Rabbit A (2·2 *Kg.*): 40 gm. of the above juice on each of two consecutive days.

Rabbit B (2·4 *Kg.*): 100 gm. of the above juice on 23.9.37. Within two hours after drenching the animal developed transient symptoms of general weakness.

24.9.37—appears normal. It received another 100 gm. of the above juice.

Result: Negative.

Some of the bulbs were minced and dried and drenched to rabbits as follows:—

Rabbit C (2·2 *Kg.*): 10 gm. of the dry bulbs (= approximately 17 gm. fresh bulb) on each of two consecutive days.

Rabbit D (2·05 *Kg.*): 20 gm. of the dry bulbs on each of two consecutive days.

Result: Negative.

Sheep 44141 (*Full-mouth*; 46 *Kg.*): 400 gm. of the fresh bulbs at 9 a.m. and 400 gm. at 3 p.m. on 7.10.36.

8.10.36—8 a.m., pronounced diarrhoea; laboured respiration; not feeding; heart-action normal; temperature 104° F. Another 800 gm. of fresh bulbs in two doses.

9.10.36—8 a.m., pronounced diarrhoea; laboured respiration; accelerated and strong pulse; slight apathy; losing in condition; temperature 104° F. Another 800 gm. of the fresh bulbs in two doses.

10.10.36—Condition as on 9.10.37; temperature 103° F.

11.10.36—Condition as on 9.10.37; temperature 102° F.

12.10.36—Animal feeding; diarrhoea less severe; heart-action normal. The animal appeared in normal health again on the 16.10.36.

PAPAVERACEAE.

Argemone mexicana L.

Common names: Steekbossie; bloudissel; Bathurst burweed; Mexican poppy; prickly poppy; Texas poppy; yellow poppy; devils fig. Erroneously called " Scotch Thistle ".

Origin: Cultivated lands, Onderstepoort, Transvaal.

State and stage of development: Fresh and in the pre-flowering stage.

Sheep 43795 (*Full-mouth*; 45 *Kg.*): 300 gm. of the fresh plant on each of three consecutive days.

Sheep 44141 (*Full-mouth*; 46 *Kg.*): 800 gm. of the fresh plant on each of three consecutive days. Total amount of plant drenched 2,400 gm.

Result: Negative.

Seddon and Carne (1927) report that the plant is not toxic to sheep. They fed 4 lb. of the green leaves to a sheep in twelve days and 1 lb. of the green fruiting heads to another sheep in eight days with negative results. Furthermore a sheep, which was drenched with crude aqueous extracts of the green leaves and fruiting heads (2 lb. equivalent) suffered no ill-effects.

POLYGONACEAE.

Rumex ecklonianus Meisn. (O.P.H. No. 8730; 8.12.36).

Common names: Tongblaar, dock.

Origin: Rooispruit, Cape Province.

State and Stage of development: Dry and in the flowering stage.

Rabbit A (1·8 *Kg.*): 10 gm. of the dry plant on each of eight consecutive days.

Rabbit B (2·3 *Kg.*): 20 gm. of the dry plant on each of eight consecutive days. The total amount of dry plant drenched was 160 gm.

Result: Negative.

SUMMARY.

Chemical tests and drenching and feeding experiments were conducted with fourteen different plants.

It is significant that hydrocyanic acid was detected in specimens of wilted *Medicago sativa* L. (lucerne), *Tephrosia macropoda* E. Mey (fish poison); and *Atriplex semibaccata* R.Br. (creeping salt bush), whilst none was detected in fresh specimens of these plants. The possibility of hydrocyanic acid poisoning through the eating of these plants in the wilted state must, therefore, be considered.

No hydrocyanic acid was detectable in fresh specimens of *Tephrosia Vogelii* Hook.

Acalypha indica L. is a deadly poison, the active principles being hydrocyanic acid (cyanogenetic glucoside) and a substance causing intense dark chocolate-brown discolouration of the blood, and gastrointestinal irritation.

Large amounts of bulbs of an *Urginea* sp. (O.P.H. No. 5603; 23.9.36) caused transient symptoms of poisoning in a sheep.

The results of feeding and drenching experiments conducted with the following plants were negative:—

Crassula expansa Ait.
Melica decumbens Thunb.
Canavalia ensiformis DC.
Ornithogalum Pretoriense Bkr.
Argemone Mexicana L.
Rumex ecklonianus Meisn.

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The Distribution and Possible Translocation of Icterogenin in *Lippia rehmanni* (Pears).

By G. C. S. ROETS, Section of Toxicological Chemistry, Onderstepoort.

It has been definitely established for a number of plants that the concentrations of their active toxic principles in their various parts differ to a remarkable degree.

By drenching 500 gm. amounts of dried powdered leaves of *Lippia rehmanni* (Pears) mixed with water to sheep through a stomach tube, Quin (1933) found that the plant causes geeldikkop symptoms. The active principle has been isolated in crystalline form by Rimington and Quin (1935). On account of the marked icterus this substance produces, it was proposed to call it " Icterogenin ". A method was devised by Rimington, Quin and Roets (1937), which enabled the author to carry out with a fair degree of accuracy quantitative determinations of Icterogenin in the various organs of the plant.

When, on one occasion, *Lippia rehmanni* (Pears) material was being collected on a plot established at Onderstepoort, the characteristic root system aroused attention. Roots were found up to 12 feet in length and ¾-inch in diameter. The relatively thick cortex could easily be removed from the stele by hand, when fresh. A sample of these roots was taken on the 6th April. 1936. It was air-dried and powdered and a quantitative determination of Icterogenin was carried out. It was found that 350 gm. yielded 2·5 gm. of the sodium salt of Icterogenin, equivalent to 0·71 per cent.

This was a very high figure, since from previous batches of dried leaves 0·25 per cent. had been considered an excellent yield. The available plants, which were in the post-seeding stage by that time, were dug up, divided into leaves, stems, root cortex and root steles, and dried in the laboratory by spreading on large sheets of paper. Quantitative determinations were carried out separately on a powdered sample of each fraction, and the yields expressed as the percentage sodium salt of Icterogenin, were as follows:—

Leaves	0·096
Stems	a trace only.
Root cortex	2·04
Root steles	a trace only.

These figures were so suggestive that it was decided to carry out further investigations on *Lippia rehmanni* (Pears). An evenly distributed patch of plants, growing under natural conditions on the farm Derdepoort near Pretoria, was selected for this purpose, and material gathered from time to time. Determinations were made on the root cortex and leaves, collected from the same plants, representing respectively the underground and above ground localisation of the toxic substance. The inflorescences were included with the leaves because the amounts of the former were relatively very small.

The data obtained during the period from the 6th May, 1936 to the 27th April, 1937, are tabulated below. The rainfall figures quoted were recorded at Onderstepoort approximately 4 miles from the spot where the plant material was collected, the rainfall in both places being approximately the same.

The figures for the leaves and root cortex are expressed as the percentage of sodium salt of Icterogenin yielded by air dried materials. The percentage of sodium in the sodium salt is about 4·1. Only traces of Icterogenin could be detected in the stems and steles of the roots.

	Date of sampling.	Percentage sodium salt of Icterogenin.		Rainfall in inches.	Remarks.
		leaves.	Root cortex.		
1936.					
March........	—	—	—	8·50	—
April.........	—	—	—	0·51	—
May..........	6	0·17	1·59	4·26	Post seeding stage.
June.........	—	—	—	—	—
July..........	—	—	—	—	—
August.......	—	—	—	—	—
September....	28	0·28	1·01	0·46	Budding stage.
October.......	—	—	—	2·68	—
November....	5	0·12	1·76	6·53	—
December.....	10	0·10	3·38	3·37	—
1937.					
January......	—	—	—	4·51	—
February.....	—	—	—	10·64	Rain nearly all in first half of month.
March........	11	0·32	2·54	1·38	Post-seeding stage; some plants pruned.
April.........	27	0·26	1·92	2·20	Post-seeding stage; from unpruned plants.
April.........	27	0·96	4·40	—	Tender young leaves from pruned plants.

Appreciation of the significance of the above data will be facilitated by recalling certain aspects of plant poisoning as it occurs in the field.

With rare exceptions like *Cicuta occidentalis*, which occurs in parts of the United States of America and of which the old rootstocks when exposed through heavy rains are exceedingly poisonous throughout the year (Howes, 1933), plant poisoning in stock is, in nearly every instance, caused by the above ground parts of plants, which are ingested by the animals while grazing. Animals may eat the plants accidentally or be attracted by the fresh greenness of the leaves, whilst the remaining vegetation is still dry, as for example occurs in the case of " tulp " poisoning caused by species of *Homeria* and *Moraea* (Steyn, 1928) and " gifblaar " poisoning caused by *Dichapetalum cymosum*. Plants which have a high nutritive value and are eagerly eaten by stock may occasionally become poisonous and be responsible for heavy losses. As an example, certain species of *Geigeria* may be mentioned, normally good fodder plants, but at times becoming highly toxic. From these plants which are the cause of " vermeersiekte " (vomiting disease) in sheep, Rimington, Roets and Steyn (1936) have isolated the active principle " vermeeric acid ". Hydrocyanic acid poisoning may frequently occur in animals grazing on natural pastures on which certain grasses (species of *Cynodon*, *Sorghum*, etc.), are present. These grasses under certain sets of conditions are liable to develop dangerous quantities of HCN. Geeldikkop may be caused in an analogous manner at certain times by species of *Tribulus* or *Panicum*.

Returning to the experimental findings quoted in Table I. it is clear that wide fluctuations may be encountered in the toxicity of *Lippia rehmanni* (Pears). The plants which are not disturbed by grazing or cutting show periodical variations between 0·10 and 0·32 per cent. in the concentration of the toxic substance. As the soil in the selected patch showed a high degree of uniformity, the variations in poison concentration may be due either to the stage of growth or to the metabolic activities of the tissues as influenced by climatic conditions.

Stage of growth, which has received due consideration from modern workers on the nutritive value of pasture plants, is of great importance also as far as the toxicity of plants is concerned. Thus *Echinopogon ovatus* Beauv., the " Noogoora Burr ". is poisonous only in the young stage while still bearing its cotyledons. *Lotus australis* Andr., when immature, contains a cyanogenetic glucoside but none of this toxic substance when mature (Howes, 1933). The leaves and stems of *Cicuta occidentalis* are poisonous when young, but harmless in the later stages of development. At the period of reproduction the nicotine concentration in the leaves of the tobacco plant increases (Vickery, Pucher and Levenworth, 1933). In *Lippia rehmanni* there is a decrease from 0·28 in September to 0·10 in December in the Icterogenin content of the leaves. There is, however, a rise to 0·32 in March, 1937, followed by a decrease to 0·26 in April, a month later. Plants in the post-seeding stage on the 6th May, 1936, yielded 0·17, while the concentration in the sample almost a year later (27th April, 1937) was 0·26, the plants again being in the post-seeding stage.

From the above figures, it appears that the stage of growth does not determine the toxicity in the case of *Lippia rehmanni* (Pears).

The fact that many plants store poisonous substances in their underground parts was well known to the Natives in many countries before Civilisation. Thus bulbs of *Adenium lugardi* form one of the sources of Bushman arrow poisons. Also in *Glyceria spectabilis* the hydrocyanic compound is distributed in the panicles, leaves, and roots (Minssen, 1934). Guerin (1933) pointed out that all the various organs, with the exception of fruits, of *Glyceria aquatica* contain a cyanogenetic glucoside. Marais and Rimington (1934) determined quantitatively the hydrocyanic acid liberated by " Linamarin " in different parts of *Dimorphotheca cuneata* Less., and found it to be distributed in the flowers, leaves, stems and roots. In *Urginea Burkei* Baker the flower heads, leaves and bulbs are poisonous (Steyn, 1934). According to information which was kindly given to me by Dr. D. G. Steyn, of Onderstepoort the minimal lethal dose for rabbits of the fresh leaves of *Dichapetalum cymosum*, collected on the same day varied from 1·2 to 6 gm. per kilo body weight. A dose of 60 gm. of thte underground stems of the same plants proved harmless, whereas in previous experiments the cortex of the underground stems had been far more toxic than the leaves.

Lippia rehmanni is no exception to the numerous examples recorded, where toxic substances are distributed both in the underground and above-ground parts of the plants. In comparing the figures tabulated above it is, however, of interest to note that a decrease in the Icterogenin content of the leaves is accompanied by a corresponding increase in that of the root cortex from September to December. Conversely in the March sample there is a decrease in the Icterogenin content of the root cortex and an increase in the leaves. In April the concentrations decreased both in the leaves and in the root cortex. The sample (27th April), which had been defoliated by cutting and pruning on the 11th March, so as to simulate the effect of trampling and close grazing, showed a markedly higher Icterogenin concentration, both in the leaves and in the root cortex, as compared with that of the undisturbed plants collected on the same day. These figures strongly suggest a translocation between the upper and underground parts of the plant. A consideration of the data on the pruned plants indicated a definite possibility that pruning, resulting in fresh developmental activity, stimulates the plants to synthesize an excessive amount of Icterogenin, which thereafter may be relatively rapidly transferred to the root cortex.

In this connection it may be mentioned that on Karoo pastures geeldikkop as caused by *Tribulus* may sometimes occur when the plants are well developed, although it is more frequently encountered on closely grazed and trampled pastures. The same applies to " geeldikkop " caused by veld grass. Steyn (1928) observed the disease on old lands. *Panicum maximum* was suspected of causing these outbreaks. Rimington and Quin also report cases of geeldikkop on closely cut grass pasture, on which *Panicum laevifolium* predominated. So far attempts to detect Icterogenin in these plants have failed, although work along these lines is still in progress.

Apart from pruning and cutting, climatic conditions may be responsible for the rate of Icterogenin synthesis and its possible translocation in the plant. Thus Henrici (1926) found that in certain grasses hydrocyanic acid appears suddenly, only to disappear again a few hours after a heavy rain, when the wilted tissues recover normal turgescence. Climatic conditions are probably responsible for, the development of toxic amounts of hydrocyanic acid in *Cynodon incompletus* Nees (Finnemore and Jaffray, 1935). *Atalaya hemiglauca* F. Muell. causes the disease known as "walkabout" in Australia. In many cases the plants develop little or none of the responsible saponin under the less favourable Northern and Southern temperature conditions (Howes, 1933). Sunshine and temperature may influence the alkaloidal content of *Atropa belladonna*. In conditions of low temperature and deficient sunshine the plant was found to be lower in alkaloidal content. Moreover the type of light may influence the development of the toxic substance in *Datura stramonium* L. (Steyn, 1934). Ghosh and Krishna found the Ephedrin content of *Ephedra* lowest during the rainy season and highest under the drier climatic conditions in autumn. In *Lippia rehmanni*, taking into consideration the unpruned plants only, the highest figure for the root cortex and the lowest one for the leaves were both found in the sample collected on the 10th December after a fair, early summer rainfall. Root cortex gathered on the 28th September after only a few spring showers had followed the dry winter months, gave the lowest yields of Icterogenin so far recorded. The concentration was the highest in the leaves after a high followed by a moderately low rainfall. Further work may throw more light on this behaviour of the plant.

Icterogenin occurs in the plant as the free acid. Hand sections have been made through various tissues rich in Icterogenin but no crystalline material could be observed microscopically. It is evidently associated with other organic substances, which may influence its physical behaviour. The sodium salt of Icterogenin, for example, while still accompanied by other organic substances, is fairly soluble in water, as was evident during the process of isolation. It is, however, almost insoluble in water when pure.

SUMMARY.

(1) Icterogenin may be present in variable amounts in the leaves and inflorescence of *Lippia rehmanni* (Pears) whereas only traces are to be found in the stem and root stele.

(2) There is a possible translocation of the toxic substance to the root cortex from the leaves, where presumably it is formed. The further possibility exists that the Icterogenin so stored in the root cortex may under certain specific conditions be translocated to the leaves of the plant. This point is receiving special attention in further work on this problem.

(3) The stage of growth does not appear to be of great importance as far as the toxicity of *Lippia rehmanni* (Pears) is concerned.

(4) Climatic conditions may to a large extent influence the synthesis and translocation within the plant tissues.

(5) Pruning and cutting cause a marked increase in the concentration of Icterogenin in both the leaves and root cortex.

(6) From the work carried out on the plant *Lippia rehmanni* (Pears) as described in this paper, the suggestion presents itself that

the reciprocal translocation of toxic principles between the above-ground and the subterranean portions of a plant may be a factor of great significance in the determination of its toxicity. This point merits further investigation.

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Section IV.

Toxicology.

STEYN, D. G. ... ... The toxicity of oil of turpentine for domestic animals 591

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The Toxicity of Oil of Turpentine for Domestic Animals.

By DOUW G. STEYN, Section of Pharmacology and Toxicology, Onderstepoort.

I. INTRODUCTION.

As a number of cases of suspected poisoning in stock with oil of turpentine has been reported to us in the course of the last few years the information in this article may be of value to those concerned.

Latterly serious mortality occurred in horses and donkeys which had been drenched by means of a bottle with the following mixture:—

" 4 oz. of oil of turpentine,
1 drachm of extract of male fern,
1 pint of raw linseed oil."

This mixture was prescribed by one of our Government Veterinary Officers and the owner of the animals alleged that the quantity of oil of turpentine is excessive and had caused the mortality among his animals. Half-doses were prescribed for young animals.

II. NATURE OF OIL OF TURPENTINE.

Oil of turpentine is a colourless, clear liquid with a peculiar odour and a pungent, somewhat bitter, taste obtained by distillation and rectification from turpentine.

Turpentine is an oleo-resin obtained from various species of *Pinus* growing in different parts of the world.

Oil of turpentine contains various hydrocarbons (terpenes), like carene, pinene ($C_{10}H_{16}$) and camphene, and resin acids.

" Aleppo turpentine " is obtained from *Pinus halepensis.*

" Bordeaux turpentine " is obtained from *Pinus maritima.* It is known as " galipot ".

" Canada turpentine " is also obtained from *P. maritima.*

" Carpathian turpentine " is obtained from *Pinus cembra.*

"Chian turpentine" is obtained from the Mediterranean tree, *Pistacia terebinthus*.

"Common or white turpentine" is obtained from different species of *Pinus*.

"Hungarian turpentine" is obtained from *Pinus pumilio*.

"Larch or Venice turpentine" is obtained from the larch tree, *Larix europea*. It is a viscid liquid of a yellowish or yellowish-green colour.

"Strassburg turpentine" is obtained from the European spruce or fir tree, *Abies pectinata*.

It should be mentioned that in commerce there is a *turpentine substitute*, called "white spirit", with a boiling range from 140° to 220° F., whilst true oil of turpentine boils at about 312·8° F. "White spirit" is a distillation product of petroleum and is probably more poisonous than oil of turpentine, as it is more volatile.

Petroleum, resin oil and wood turpentine are the most common adulterants found in oil of turpentine. Wood turpentine is obtained by distilling the roots and stumps of various species of *Pinus*.

Furthermore petroleum, paraffin oils, rosin, rosin oil, petroleum benzin, kerosene oil and similar hydrocarbons may occur as impurities in oil of turpentine.

Oil of turpentine should be stored in well-closed containers (preferably dark-coloured ones) in a cool and dark place. In light and in the presence of oxygen oxidation processes cause the formation of formic, acetic and camphoric acids, camphoric aldehyde, and hydrogen peroxide.

Oil of turpentine should have a residue of not more than 0·5 per cent. when evaporated quickly in a flat dish over a water-bath.

[British Pharmac. Codex (1934); Dorland (1923), Squire (1916).]

III. Toxic Doses.

The toxic doses of oil of turpentine vary according to its origin, chemical constitution, and storage. The more volatile the constituents and the higher the degree of oxidation of these constituents the more poisonous the oil of turpentine will be. The presence of impurities like paraffin oils, petroleum benzin, kerosene oil and other volatile hydrocarbons renders oil of turpentine more toxic.

Fröhner (1919) states that horses and cattle tolerate single doses of 250 to 400 grams (= ±8 to 13 oz.) of oil of turpentine, whilst amounts of from 500 to 1,000 grams (= ±17 to 33 oz.) induce colic, diarrhoea and haematuria in horses. He saw a pronounced haemorrhagic nephritis in a horse which had received 500 grams of oil of turpentine. The animal, however, recovered. Hertwig (Fröhner, 1919) reports that dogs, which had received from 8 to 30 grams (= ±¼ to 1 oz.) of oil of turpentine died from gastro-enteritis.

A child died about fifteen hours after having swallowed 15 grams of oil of turpentine (Lewin, 1929). Leschke (1932) states that 10 to 15 c.c. of oil of turpentine is poisonous for man, persumably when taken undiluted. The British Pharmaceutical Codex, 1934, prescribes 8 to 16 c.c. (=2 to 4 fluid drachms) of oil of turpentine as an anthelmintic for man.

Dierschke (1935) accidently injected three horses weighing approximately 700 Kg. intravenously with 15 c.c. of oil of turpentine. Each injection was completed in fifteen seconds. Within a few minutes after injection two of the horses developed the following symptoms of poisoning: restlessness, shaking the head, perspiration, accelerated pulse and respiration, one animal developed an irregular pulse and did not feed. The temperatures recorded were from 39·4 to 40·0° C. The third horse also showed similar symptoms within a few hours after the injection. The following morning the animals appeared normal again.

Jeckowitsch (Dierschke, 1935) injected one hundred horses suffering from laryngo-pharyngitis intravenously with 3·0 c.c. of oil of turpentine with very satisfactory results. Horses, which had received 2·0 c.c. of oil of turpentine intravenously five times at intervals of from four to six days developed no symptoms of poisoning [Scharungowitsch (Dierschke, 1935)].

Dierschke suggests that experiments be conducted in order to ascertain the toxic doses of oil of turpentine when injected intravenously as this method of administering this aetherial oil may prove of great benefit in diseases of the respiratory tract.

When injected subcutaneously oil of turpentine causes the formation of sterile abscesses.

ONDERSTEPOORT EXPERIMENTS.

As mixtures of oil of turpentine, extract of male fern and raw linseed oil are commonly used as anthelmintics in horses it was thought advisable to administer both oil of turpentine and extract of male fern in the same mixture in order to ascertain whether they act synergistically and increase each others toxicity.

The oil of turpentine used in these experiments is that prepared by Riedel and de Häen, Hannover, Germany, except where stated otherwise.

The extract of male fern used bears the following label: " Extract Ethere de Fougére Mâle Vétérinaire, Titre 24-25 per cent. filicme brute. Gignoux Freres & Cie, Lyon ".

The raw linseed oil was the " Genuine Raw Linseed Oil, Thistle Brand " obtained from W. McIntosh, 473 Church Stieet, Pretoria.

All the horses were starved for approximately eighteen hours before being drenched; they, however, had free access to water.

ONDERSTEPOORT EXPERIMENTS.

Animal.	Age and condition.	Weight.	Quantity of oil of turpentine administered per stomach tube.	Result.
Rabbit A....	Full-grown and in good condition	2·0 kg.	4·0 c.c. of oil of turpentine* in 33 c.c. of raw linseed oil	Not feeding well for ± 36 hours after drenching and slight diarrhoea set in ± 24 hours after drenching and lasted approximately 12 hours. Thirty-six hours after administration of the oil of turpentine the animal was normal again.
Rabbit B....	Full-grown and in good condition	2·25 kg.	8·0 c.c. of oil of turpentine* in 50 c.c. of raw linseed oil	Severe diarrhoea set in within 12 hours after drenching and lasted for four days. During this period the animal did not feed, was very apathetic, and lost in condition. Respiration and pulse were accelerated. Diuresis was present. Recovery took place five days after drenching.
Horse 21413	Gelding in fairly poor condition	—	(1) 120 c.c. (= 4 oz.) of oil of turpentine* and 4·0 c.c. (= 1 drachm) of extract of male fern in 600 c.c. (= 1 pint) of raw linseed oil on the 25/8/36	The animal suffered no ill-effects.
			(2) 120 c.c. of oil of turpentine and 8·0 c.c. of extract of male fern in 600 c.c. of raw linseed oil on 16/11/36	The animal suffered no ill-effects.
			(3) 240 c.c. of oil of turpentine and 8·0 c.c. of extract of male fern in 600 c.c. of raw linseed oil on 15/12/36	On the 16/12/36 and 17/12/36 the animal passed partly undigested soft faeces and was not feeding well. On the 18/12/36 diarrhoea and diuresis were present, but the animal appeared quite healthy and was feeding well. On the 19/12/36 the faeces were normal again.

ONDERSTEPOORT EXPERIMENTS—(*continued*).

Animal.	Age and condition.	Weight.	Quantity of oil of turpentine administered per stomach tube.	Result.
Horse 21357	Aged gelding in fair condition	—	(1) 120 c.c. of oil of turpentine and 4·0 c.c. of extract of male fern in 600 c.c. of raw linseed oil on 16/11/36 (2) 120 c.c. of oil of turpentine and 4·0 c.c. of extract of male fern in 600 c.c. of raw linseed oil on 15/12/36	The only noticeable symptoms were that the animal was off its feed for about three hours and the faeces slightly soft in consistence during the day after drenching. Fairly severe diarrhoea, diuresis and loss of appetite, lasting for about two days, set in about twenty-four hours after drenching. Complete recovery had taken place on the fourth day after drenching.
Horse 21358	Aged mare in fairly good condition	—	(1) 120 c.c. of oil of turpentine and 4·0 c.c. of extract of male fern in 600 c.c. of raw linseed oil on 16/11/36	Suffered no ill-effects whatsoever.
			(2) 240 c.c. of oil of turpentine and 4·0 c.c. of extract of male fern in 600 c.c. of raw linseed oil on 15/12/36	Results similar to that described under (2) of Horse 21357.
Horse 21415	Aged stallion in poor condition	—	120 c.c. of oil of turpentine and 8·0 c.c. of extract of male fern in 600 c.c. of raw linseed oil on 16/11/37	Suffered no ill-effects whatsoever.

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* Prepared by Merck, Darmstadt, Germany.

From the foregoing experiments it is evident that 8·0 c.c. of oil of turpentine administered in 50 c.c. of raw linseed oil induced transient symptoms of poisoning in a rabbit, while 4·0 c.c. administered in 30 c.c. of raw linseed oil caused a slight diarrhoea and temporary loss of appetite.

It would appear that 120 c.c. (=4 oz.) of oil of turpentine and 4 c.c. (=1 drachm) of extract of male fern administered in 600 c.c. (=1 pint) of raw linseed oil is a safe mixture for full-grown horses in fairly good condition. Horse 21413 even tolerated twice this quantity of oil of turpentine and extract of male fern without showing definite symptoms of poisoning. In view of the fact, however, that horse 21357 reacted fairly severely to a mixture of 120 c.c. of oil of turpentine and 4 c.c. of extract of male fern in 600 c.c. of raw linseed oil it would not be advisable to administer larger quantities than these of oil of turpentine and extract of male fern to full-grown horses in fairly good condition. Horses in poor condition and young horses should receive half of the above doses, or less, according to age, size and condition. The prescribed dose of 120 c.c. of oil of turpentine is that for the pure, unoxidised and unadulterated product. The oxidised and adulterated oil containing kerosene oil, benzine, paraffin, etc., will probably be more toxic.

IV. Symptoms of Poisoning.

Oil of turpentine is a severe irritant to the skin and mucous membranes. It is, therefore, obvious that it should not be administered as such *per os* but be mixed with demulcents and emollients. As a rule it is administered in raw linseed oil. It is known to have caused immediate death when administered as such *per os*, death being due to choking owing to the irritant nature of the oil.

Given internally in toxic doses it causes stomatitis, laryngitis, pharyngitis, and acute catarrhal gastro-enteritis.

It is excreted to a large extent by the lungs and kidneys. In acute poisoning the exhaled air smells of the oil. Large doses of oil of turpentine cause an acute nephritis, which may be haemorrhagic, and also pyelitis and cystitis.

The central nervous system is also affected. At first there is stimulation manifested in increased reflexes, muscular tremors, spasms, excitement, palpitation of the heart, accelerated pulse and dyspnoea. These symptoms are followed by those of paralysis, namely apathy, dizziness, staggering, fall in blood-pressure, heart-weakness, general paralysis, slowing, and eventually paralysis of the respiration.

If oxidation of the oil of turpentine had occurred it may cause methaemoglobinaemia. According to Winternitz (Petri, 1930) there may be a pronounced leucocytosis in cases of poisoning with oil of turpentine.

The urine has a characteristic violet-like odour.

As in mercurial poisoning death may be a sequel to the nephritis caused by oil of turpentine.

If excessive quantities of oil of turpentine are inhaled it will cause bronchitis and pneumonia in addition to the above symptoms.

V. POST-MORTEM APPEARANCES.

In cases of acute poisoning with oil of turpentine general cyanosis, nephritis, and severe irritation of the gastro-intestinal mucosa will be seen. There is hyperaemia of all the internal organs. Oil of turpentine is detectable in the thoracic cavities and stomach contents by means of its characteristic odour.

In cases of chronic poisoning there may be nephritis and chronic gastro-enteritis.

VI. CONTRA-INDICATIONS.

Oil of turpentine should not be administered to animals suffering from congestion of the kidneys, nephritis, or gastro-enteritis.

VII. TREATMENT.

No specific antidote to oil of turpentine is known and symptomatic treatment is to be applied. If possible stomach lavage should be applied. Emollients, demulcents and astringents (barley gruel, linseed decoctions, raw linseed oil, lime water, tannic acid) and general heart stimulants (caffeine) should be administered.

In cases where paralysis has already set in central nervous system stimulants (strychnine, caffeine) and respiratory stimulants (lobeline) should be given.

VIII. SUMMARY AND CONCLUSIONS.

1. The following mixture appears to constitute no danger to full-grown horses not suffering from congestion of the kidneys, nephritis or gastro-enteritis:—

“ 120 c.c. (=4 oz.) of oil of turpentine,
4 c.c. (=1 drachm) of extract of male fern,
600 c.c. (=1 pint) of raw linseed oil ”.

As stated before young animals and animals in poor condition should receive half of this mixture, or less, and only pure unadulterated and unoxidised oil of turpentine should be used.

2. Experiments conducted at Onderstepoort with oil of turpentine and extract of male fern are described.

3. The toxic doses, symptoms of poisoning, post-mortem appearances, and treatment of cases of oil of turpentine poisoning are discussed.

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Section V.

Mineral Metabolism and Nutrition.

GROENEWALD, J. W. ... Osteofibrosis in equines 601

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Osteofibrosis in Equines.

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CASES of osteofibrosis are not infrequently reported from race-horse stables where the animals are fed on cereal concentrates, and where the hay ration is kept relatively low. Unless legume hay is fed, or a correction is made in the diet by the supplementation of calcium, rations consisting for the most part of cereals may prove to have a high phosphorus and a low calcium content.

The occurrence of osteofibrosis has been reported from various countries, but has assumed a magnitude of considerable economic importance in India and the Philippine Islands, where the native feeds supplied to animals are relatively low in calcium. Although the disease is well known, the nomenclature used by different writers in describing it, differs considerably. The result is that a certain degree of confusion exists in regard to the various terms applied to this and related osteodystrophic disorders.

An attempt was, therefore, made to produce cases of osteofibrosis in horses. The material collected would then form a useful basis of bone pathological comparisons in osteodystrophic diseases.

LITERATURE.

Theiler *et al.* (1937) describe rickets or osteomalacia as a phosphorus deficiency disease, and suggest that calcium deficiency produces osteofibrosis. Their observations were carried out mainly on cattle.

Niimi (1927) considered that a deficiency of calcium salts in the diet was the cause of osteomalacia, and actually produced what he believed to be osteomalacia, but which in reality was osteofibrosis, in two old horses. The ration given to these horses consisted of barley and contained 2·09 gms. CaO and 27·57 gms. P_2O_5.

In a subsequent trial Niimi and Aoki (1927) produced the disease in horses receiving 3·6 gms. CaO and 47·4 gms. P_2O_5 daily. The control group remained normal when receiving a ration that contained 28·0 gms. CaO and 41·6 gms. P_2O_5.

Marek (1922) points out that osteodystrophic diseases develop in pigs receiving a ration which does not show an Erdalkali-Alkalizität ($CaO + MgO - P_2O_5$ in mgm. equivalents) of 20 to 25 mgm. equivalents. This author apparently does not distinguish aetiologically between osteomalacia and osteofibrosis.

Kintner and Holt (1932) describe what they considered to be osteomalacia in horses and conclude that, " osteomalacia will develop in horses when the ratio of calcium oxide to phosphorus pentoxide is 1:2·9 ". According to the pictures of the bone pathology, these authors were dealing with osteofibrosis. It was further shown that, " the condition did not develop during the nine-month period when this ratio was 1:1·9. Evidence is presented showing that the calcium content of the drinking water was of particular importance ".

It is apparent from the work of these authors that the indigenous feeds generally given to horses in the Philippines are so low in calcium, that unless limestone is added to the ration, equines are liable to contract osteofibrosis.

The work of Sturgess (1927) definitely showed that the possible lack of the vitamins contained in greenfeed could not be considered as contributory factors in the occurrence of osteofibrosis. This worker produced the disease on a ration consisting of bran and 10 pounds of green grass, and came to the conclusion that osteoitis fibrosa was a possible deficiency of calcium, the condition being intensified when there was a co-existing excess of phosphorus.

Crawford (1927) while working in Ceylon, came to the conclusion that the determining factor in the production of osteoporosis was the proportion of calcium to phosphorus present in the diet.

Olver (1933) in describing a case of osteoporosis in an aged gelding in India, gives the predisposing cause of the disease as diet composed mainly of cereals, such as bran and oats, with inadequate fodder of leguminous nature.

It is evident from the work of Theiler (1934) that the disease which was produced by Crawford and also by Olver, was not osteoporosis but osteofibrosis.

It is quite possible, according to Mitchell (1935), that a large number of common horse ailments such as arthritis of the knees and fetlocks, may be attributed to varying degrees of osteodystrophic diseases.

It is evident from the literature on osteofibrosis, that uncertainty exists in regard to the actual causation of the disease. Certain authors hold that osteofibrosis in horses is associated with abnormal calcium metabolism. The dietary calcium may be insufficient for the requirements of the animal, or the deficiency may be accentuated by a relatively large excess of phosphorus, or both factors may operate. The result is the same, viz., that the animal is starved for calcium.

EXPERIMENTAL.

Five yearling fillies, numbers 20722, 20723, 20721, 20739 and 20745, were procured for the purpose of producing clinical symptoms of osteofibrosis.

A common basal ration consisting of 1,000 gms. crushed oats, 1,000 gms. yellow maize, 1,000 gms. wheaten bran, 400 gms. greenfeed, 500 gms. teff hay and 2 ounces of $CaCO_3$, was fed to the animals for a pre-experimental period of 8 months in order to ensure uniformity of pre-experimental treatment. The horses were then grouped and received mineral supplements as shown in Table 1:—

Table 1.

Experimental Plan.

No.	Group.	Supplement.	$CaO : P_2O_5$.	Remarks.
20723 20739	1	64 gms. Na_2HPO_4	1 : 12·06	The basal ration remained unchanged, with the exception that 2 ounces of $CaCO_3$ was omitted.
20721 20722	2	75 gms. $CaCO_3$	1·8 : 1	
20745	3	0	1 : 6·3	

The quantity of hay had necessarily to be limited to a daily intake of 500 gms. per horse in order to keep the CaO content of the ration low. The basal ration contained 370 gms. protein, 4·5 gms. CaO, 28·4 gms. P_2O_5, and a $CaO:P_2O_5$ ratio of 1:6·.

The concentrate mixture, greenfeed and mineral supplement were fed to the horses at 2 p.m. daily. The hay was supplied early in the morning, and when it had been consumed the animals were muzzled and allowed to run in an open air, cement-floored paddock until 2 p.m.

In order to avoid possible complications due to internal parasitic infection, all the animals were treated for worms periodically. Daily inspection of the health and development of clinical symptoms of the horses were made. The animals were weighed monthly, and blood was drawn for the chemical determinations of calcium, phosphorus and phosphatase. Metabolism trials were carried out during the latter four months of the experiment. In order to ensure the daily consumption of all the minerals supplied, a system of feeding was adopted whereby each horse received only as much feed as it would eat. It was necessary, however, occasionally to weigh feed back.

Rubber matting was used in the stable in order to obviate the consumption of bedding by the animals.

RESULTS.

The horses, without exception, showed osteophagia or "pica" to a marked degree, and unless muzzled, would greedily consume each other's faeces. Lack of total bulk in the ration may have contributed towards the depraved appetites shown by the animals.

The clinical progress of the disease may be given as follows:—

Horse 20723, *Group* 1. *Intake* $CaO = 4 \cdot 5$ *gms*. $P_2O_5 = 54 \cdot 29$ *gms*.

Date.	*Remarks.*
11.9.34.	Commenced supplementation of 64 gms. Na_2HPO_4 daily.
14.5.35.	A shortened stride was noticeable.
16.5.35.	Facial swellings just visible.
4.6.35.	Animal reluctant to rise in the morning.
6.6.35.	Rear feet rested alternately when standing.
11.6.35.	Facial enlargements marked, involving submaxillary bones. Horse would frequently lie down with legs outstretched. Abscess on point of left shoulder.
13.6.35.	Chewing became painful and quidding of the food was observed.
3.7.35.	Stride stiff and short, face greatly enlarged.
17.7.35.	Unable to rise, animal put into a sling.
18.7.35.	Supplement changed to 75 gms. $CaCO_3$ daily.
21.7.35.	Feed consumption improved, animal made attempts to stand.
22.7.35.	Able to walk with difficulty, thrust in off fore.
2.8.35.	Greatly recovered, kept on control ration for a year in order to observe facial swelling.

Horse 20739, *Group* 1. *Intake* $CaO = 4 \cdot 5$ *gms*. $P_2O_5 = 54 \cdot 29$ *gms*.

Date	*Remarks.*
11.9.34.	Commenced supplementation of 64 gms. Na_2HPO_4 daily.
14.5.35.	When trotted a slightly shortened stride noticeable.
21.5.35.	Ringbone appeared on near rear pastern.
28.6.35.	Stiff gait and rested rear feet alternately.
25.7.35.	Stiff, shortened stride, pasterns painful, off feed.
9.8.35.	Facial enlargement very sudden and marked.
23.8.35.	Abscess developed on near knee.
11.9.35.	Unable to rise unassisted.
13.9.35.	Fell and broke back behind sacral vertebra. Microscopical examination of bones showed osteofibrosis.

Horse 20722, *Group* 2. *Intake* $CaO=52 \cdot 29$ *gms.* $P_2O_5=28 \cdot 42$ *gms.*

Date.	*Remarks.*
11.9.34.	Commenced supplementation of 75 gms. $CaCO_3$ daily.
11.9.35.	Animal remained in good health and thrifty throughout the duration of the experiment. No facial enlargements developed, and she was retained on the same ration for an additional period of a year in order to act as further control to 20723.

Horse 20721, *Group* 2. *Intake* $CaO=52 \cdot 29$ *gms.* $P_2O_5=28 \cdot 42$ *gms.*

Date.	*Remarks.*
11.9.34.	Commenced supplementation of 75 gms. $CaCO_3$ daily.
11.9.35.	Discharged from experiment in excellent health and condition. No facial enlargements were shown.

Horse 20745, *Group* 3. *Intake* $CaO=4 \cdot 5$ *gms.* $P_2O_5=28 \cdot 42$ *gms.*

11.9.34.	Basal ration only, i.e. no mineral supplement.
16.5.35.	Slight swelling of left sub-maxillary bone.
12.7.35.	Enlargement of bone on left side of face pronounced, poor, coat stary and gait stiff.
12.11.35.	Discharged from experiment in poor condition.

The general condition and appearance of the fillies when at first admitted to the experiment, as well as at the conclusion of the experimental period, may be seen from the photographs of these animals given in Appendix I. As further illustrated in these photographs the facial enlargements involve mainly the submaxillary bones. The enlargements being particularly clear in the case of 20723, and remained unchanged after this animal received a calcium carbonate supplement for a year. The poor condition of 20723 and 20739 is apparent at the conclusion of the experiment. The characteristic way in which 20739 draws up her hind leg, and the slightly protruding tongue, is worthy of note.

The curves indicating the weights of the fillies are given in Appendix 2, and indicate clearly that only in the case of the control group, 20721 and 20722, was there any gain in weight during the experimental period. The consistent loss in weight of 20723 was checked when calcium carbonate was supplied in the ration.

Feed consumption may be regarded as satisfactory. Animals only went off feed when exhibiting severe clinical symptoms of osteofibrosis.

The figures indicating chemical blood determinations that were made are given in Appendix 3, and show no marked deviation in any particular group or constituent. It may, therefore, be concluded that blood analyses will give no assistance in the diagnoses of the disease. No indication of anaemia could be registered from the haemoglobin determinations made when the animals in group 1 were in the concluding stages of the disease.

The temperatures were recorded in every case throughout the experimental period and were not in any way affected by the progress of the disease.

The actual mineral intake of each horse is given in Table 2.

TABLE 2.

The Ca and P supplied to the horses.

Number.	P intake.	Supp.	Total P.	Ca intake.	Supp.	Total Ca.
20723......	12·41	11·3	23·71	3·41	0	3·41
20739......	12·41	11·3	23·71	3·29	0	3·29
20721......	12·41	0	12·41	3·35	34·0	37·35
20722......	12·41	0	12·41	3·17	34·0	37·17
20745......	12·41	0	12·41	3·23	0	3·23

Metabolism trials were carried out at monthly intervals during the last four months of the experiment. The results of these trials are given in Tables 3 and 4.

The quantity of calcium supplemented to horses 20721 and 20722 had necessarily to be sufficient to enable a $CaO:P_2O_5$ of 1·8:1 to be obtained. It does not, therefore, have any bearing upon the calcium requirement of horses.

TABLE 3.

Calcium Metabolism.

Date.	Number.	Group.	*Ca intake.	Ca output.		Balance.
				Faeces.	Urine.	
6/35.......	20723	1	3·41	7·49	0·88	− 4·19
	20739		3·29	5·51	0·72	− 2·94
	20721	2	37·35	23·32	2·93	+ 11·10
	20722		37·17	20·60	4·48	+ 12·09
	20745	3	3·23	5·44	0·38	− 2·59
7/35.......	20723	1	2·70	7·12	0·44	− 4·86
	20739		2·68	5·88	0·53	− 4·31
	20721	2	37·35	30·40	2·17	+ 4·72
	20722		30·24	24·00	4·30	+ 1·94
	20745	3	2·67	5·43	0·64	− 3·40
8/35.......	20723	1	37·94	14·20	0·48	+ 22·26
	20739		2·66	3·18	0·51	− 1·03
	20721	2	37·42	30·40	3·36	+ 3·66
	20722		31·28	24·20	4·60	+ 2·48
	20745	3	2·65	5·66	0·87	− 3·88
9/35.......	20723	1	37·96	27·70	2·95	+ 7·29
	20739		2·60	3·82	0·62	− 1·80
	20721	2	37·41	31·40	2·73	+ 3·28
	20722		33·36	27·40	5·04	+ 5·52
	20745	3	2·64	3·80	0·75	− 1·91

TABLE 4.

Phosphorus Metabolism.

Date.	Number.	Group.	P intake.	P output.		Balance.
				Faeces.	Urine.	
6/35.......	20723	1	23·71	7·72	4·53	+ 11·46
	20739		23·71	6·60	4·34	+ 12·77
	20721	2	12·41	10·15	0·11	+ 2·16
	20722		12·41	9·02	0·22	+ 3·17
	20745	3	12·41	5·06	0·80	+ 6·55
7/35.......	20723	1	19·60	11·20	2·42	+ 5·98
	20739		16·47	7·56	2·70	+ 6·21
	20721	2	12·41	12·10	0·06	+ 0·21
	20722		10·68	7·62	0·06	+ 3·00
	20745	3	6·31	4·47	1·54	+ 0·30
8/35.......	20723	1	16·46	7·30	1·05	+ 8·11
	20739		10·40	5·39	1·51	+ 3·50
	20721	2	12·41	9·70	0·03	+ 2·68
	20722		12·41	6·95	0·06	+ 5·40
	20745	3	8·32	5·23	0·91	+ 2·18
9/35.......	20723	1	16·53	10·48	0·17	+ 5·88
	20739		16·62	8·41	1·78	+ 6·33
	20721	2	12·41	9·60	0·08	+ 2·74
	20722		12·41	8·51	0·10	+ 3·80
	20745	3	6·82	5·00	1·68	+ 0·14

The slight variations in Ca and P intakes for different metabolism periods may be attributed to the lowered food consumption of the animal concerned. A total chemical analysis of the ration prior to each metabolism trial similarly discloses slight variations in the Ca and P contents.

It may be seen from the metabolism figures that a positive phosphorus balance was retained in all cases. The retention of phosphorus was greater in 20723 and 20739, where P intake was highest. Where the calcium was deficient in the ration, groups 1 and 3, a negative calcium balance was recorded. The retention of Ca was greatly increased immediately after Ca had been supplied to 20723, as is evident from a comparison of period 8/35 in Table 3, with the Ca retentions of 20723 for the previous periods.

A record of the X-ray photographs taken of the metacarpal bones two months prior to the conclusion of the experiment, are given in Appendix 4. The lesser defined lines are clearly shown in the metacarpal bone pictures of 20723, 20739 and 20745, as compared to the distinctly denser bones of 20721 and 20722.

DISCUSSION.

Clinical symptoms of osteofibrosis were produced in both horses, 20723 and 20739 fed a ration containing 4·5 grams CaO and 54·29 grams P_2O_5, i.e. a ratio of 1:12·06.

The disease was produced to a lesser degree in horse 20745 receiving in its ration 4·5 grams CaO and 28·4 grams P_2O_5, i.e. a ratio of 1:6·3.

In these horses, kept under laboratory conditions where no work was performed, the first clinical symptoms of osteofibrosis were seen eight months after the commencement of the experiment. When once clinical symptoms became clearly established, the disease took its course in an alarmingly rapid fashion. At first facial swellings became noticeable, these involved mainly the submaxillary bones, the enlargement of which became most marked during the last month of the experiment. There was also a thickening of the jaw bones, which became especially marked in the case of 20723.

Both horses 20723 and 20739 displayed characteristically shortened strides, the rear pasterns became painful and the animals would draw up the rear feet alternately as shown by 20739 in Appendix 1. Apparently 20723 had difficulty, or pain, in properly masticating its feed, as quidding of partly masticated hay was frequently seen in the manger.

Bone sections were taken for histopathological examination according to the technique described by Thomas and van der Wath (1937). Although this method of bone sampling was devised after the conclusion of the experiment, sections were nevertheless taken from 20723 and 20745 on the 3.11.36 and 5.11.36 respectively. The rib sections in both cases showed that osteofibrosis had been present. When horse 20739 had to be destroyed on the 13.9.35, microscopical examination of the bones left no doubt as to the presence of osteofibrosis.

The weight curves shown in Appendix 2 indicate clearly that only for the animals in group 2 (20721 and 20722), was there a steady gain in weight recorded throughout the experimental period. In group 1, horse 20739 showed a satisfactory gain in weight for seven months on the experimental ration. During the last three months, however, a rapid loss in weight occurred. A steady loss in weight was recorded in the case of 20723 and 20745.

From the figures available for blood analyses which are given in Appendix 3, it would appear that a calcium deficiency, as in 20723, 20739 and 20745, was not reflected by a lowered blood calcium during 1935. The inorganic phosphorus, phosphatase, and haemoglobin content of the blood and serum remained normal in all cases. Blood analysis is not, therefore, considered a reliable guide for diagnostic purposes in the case of osteofibrosis suspects.

An analysis of the results of the balance trials given in Table 3 shows that horses 20723, 20739 and 20745, remained on a negative calcium balance while on a daily average Ca intake of about 2·85 grams. It is evident, therefore, that the skeletons of these animals must have suffered a continual depletion of calcium salts. Such a drain upon the system must eventually lead to serious consequences.

It is significant that immediately after horse 20723 had its Ca intake raised from 2·7 gms. to 37·94 gms., the balance was 22·26 gms. as compared to −4·86 for the previous month as shown in periods 8/35 and 7/35 respectively. The demand for calcium, which a period of starvation had created, was rapidly satisfied by the sudden abundant supply, as may be seen in period 9/35 when only 7·29 gms. Ca was retained.

From a study of the results of the phosphorus (P) balance trials given in Table 4, it may be seen that all the animals showed a positive phosphorus balance. The control animals 20721 and 20722 showed an average retention of phosphorus of 23 per cent. When the phosphorus intake was 23·71 gms. as is shown in trial 6/35 in the case of 20723 and 20739, the balance was 11·46 and 12·77 respectively, or about 50 per cent. The retention of phosphorus was, therefore, considerably increased when the intake was more. Subsequent periods show a lower phosphorus balance for 20723 and 20739 because food consumption became irregular during the latter three months with the result that the intake had to be reduced during the metabolism periods.

The presence of large amounts of phosphorus and its increased utilization, may be the reason for the aggravation of the disease when there is a wide $CaO:P_2O_5$ ratio, as well as a deficiency of calcium.

When horse 20723 was given a supplement of 37·94 grams Ca at the conclusion of the experimental period, its recovery was phenomenal. As a result, it was decided to continue giving the control diet to this horse in order to note whether there would be any lessening of the facial swelling. Although a remarkable gain in condition was noted, the facial enlargement did not subside, as may be seen from the photographs in Appendix 1. These pictures were taken a year later, together with those of the control horse 20722. The latter received its original control ration until discharged on the 17.2.37.

The X-ray photographs of the metacarpal bones shown in Appendix 4 illustrates clearly that the bone in the case of horses 20721 and 20722 is more compact and thicker than that of horses 20723, 20739 and 20745. The various lines of demarcation are far more clearly defined in the case of the former animals. The medullary cavity is smaller and consequently the outer shell thicker. In the latter cases there is a wider, less dense cortex which is poorly defined.

SUMMARY.

(1) Clinical symptoms of osteofibrosis were brought about, and later definitely shown to be the disease by histo-pathological examination, in three 2-year-old fillies receiving a ration which contained 4·5 grams CaO and 54·29 grams P_2O_5 in the case of two horses, and 28·42 grams P_2O_5 in the case of the other horse.

(2) The two control fillies received in their ration 52·3 grams CaO and 28·42 grams P_2O_5. The CaO intake had necessarily to be high in order to rectify the otherwise abnormal $CaO:P_2O_5$ ratio.

(3) The control animals gained in weight, whereas the horses receiving a deficiency of CaO in their diet lost weight, became emaciated and poor in condition.

(4) Balance trials showed that the skeletons in the case of those horses receiving 4·5 gms. CaO, were continually being depleted of calcium.

(5) The phosphorus retention was greatest for the horses receiving most phosphorus in their ration.

(6) From the data available it would appear that blood Ca determinations are of little value for diagnostic purposes in cases of suspected osteofibrosis.

(7) Facial enlargements, when once established by the disease, were not reduced in size by feeding a ration which was supplemented with $CaCO_3$.

ACKNOWLEDGMENT.

The author wishes to express his appreciation to Dr. J. S. Otto for the chemical analysis and to Dr. Quinlan for the X-Ray photographs.

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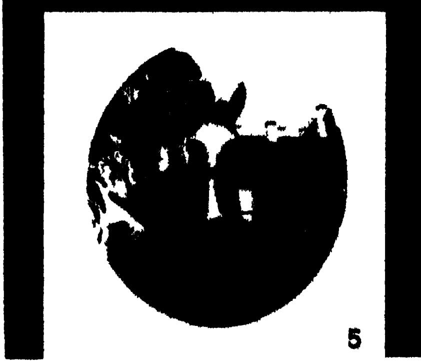
APPENDIX 1.

PHOTOGRAPHS OF HORSES.

Control horses 20721 *and* 20722 *Intake* —$CaO = 52.29$ *gm.*, P_2O_5 –28 42 *gm.*



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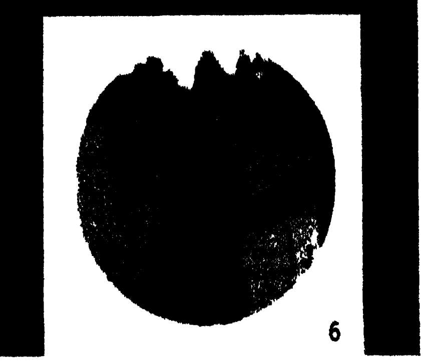

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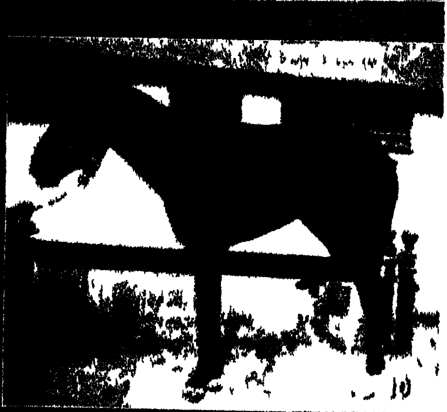
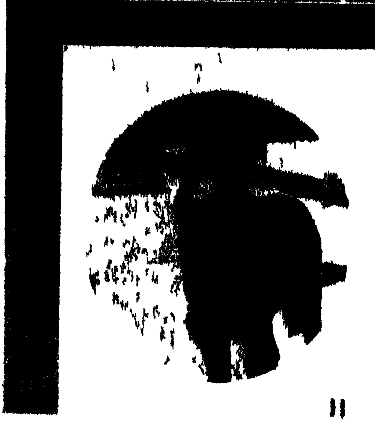
Fig. 1. Horse 20721 (29.10.34).
Fig. 3 ,, (9. 9.35).
Fig. 5. ,, (9 9.35) Face.

Fig. 2. Horse 20722 (29.10.34).
Fig. 4. ,, (9 9.35).
Fig 6. ,, (9. 9.35) Face.

Horses 20723 and 20739: Intake:—CaO=4·5 gm., P_2O_5=54 29





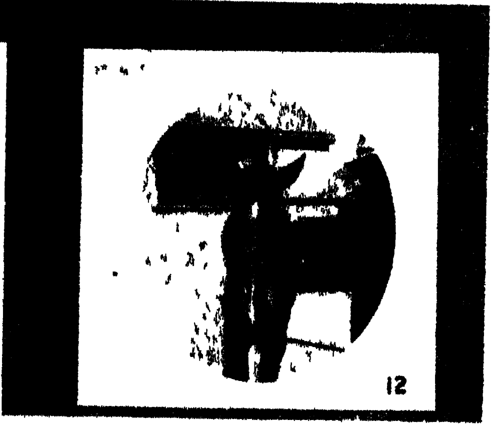


Fig. 7. Horse 20723 (29.10.34).
Fig. 9. „ (9 9.35).
Fig. 11. „ (9. 9.35)
Face.

Fig. 8. Horse 20739 (29.10 34).
Fig. 10. „ (9. 9.35).
Fig. 12. „ (9. 9.35)
Face.

Horse 20745 *Intake*—*CaO*=4.5 *gm*, *P_2O_5* =28.42 gm.

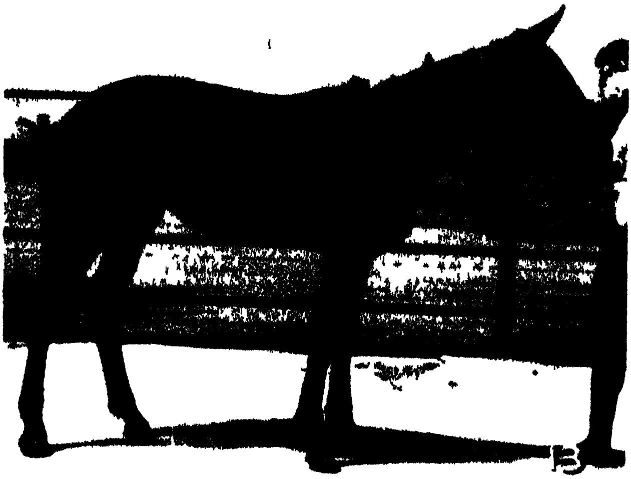


Fig 13 Horse 20745 (29.10.34)

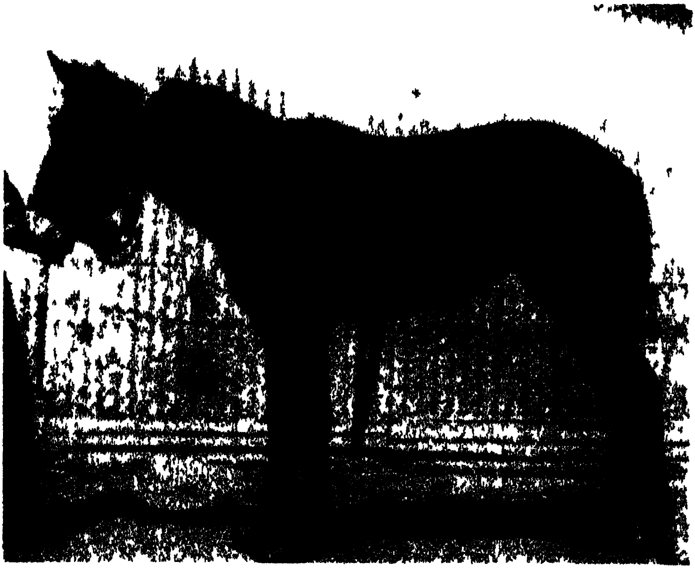


Fig 14. Horse 20745 (9. 9 35).

Horse 20745 *Intake* — *CaO*=4 5 *gm* , P_2O_5=28 42 gm.

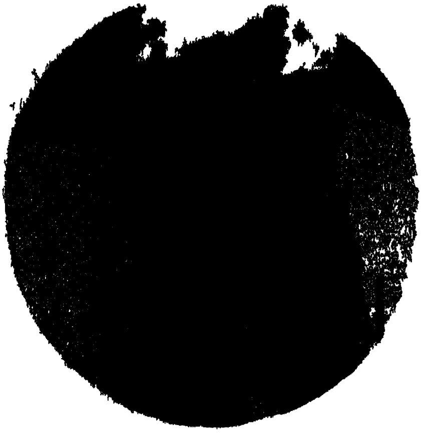


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Fig 15 Horse 20745 (9 9 35)
Face.

Horses 20722 *and* 20723 *photographed on* 17 2 37 *after receiving an intake of* 52 29 *gm* *CaO and* 28 42 *gm* *P O since the conclusion of the experiment on* 12 11 35

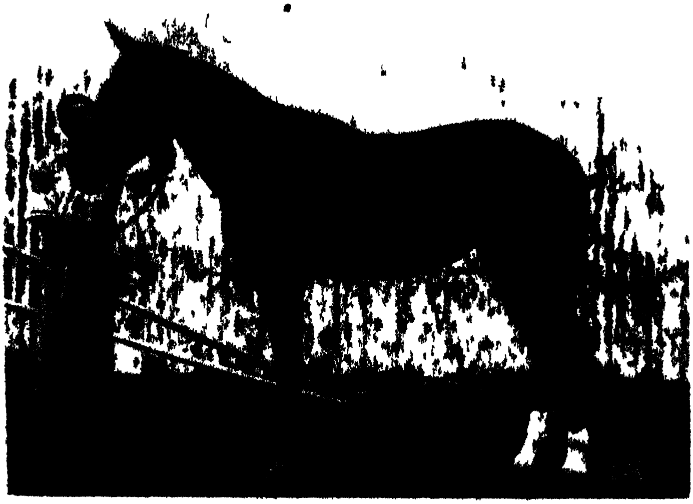


Fig 16 Horse 20722

Horses 20722 and 20723 photographed on 17.2.37 after receiving an intake of 52·29 gm. CaO and 28·42 gm. P_2O_5 since the conclusion of the experiment on 12.11.37.

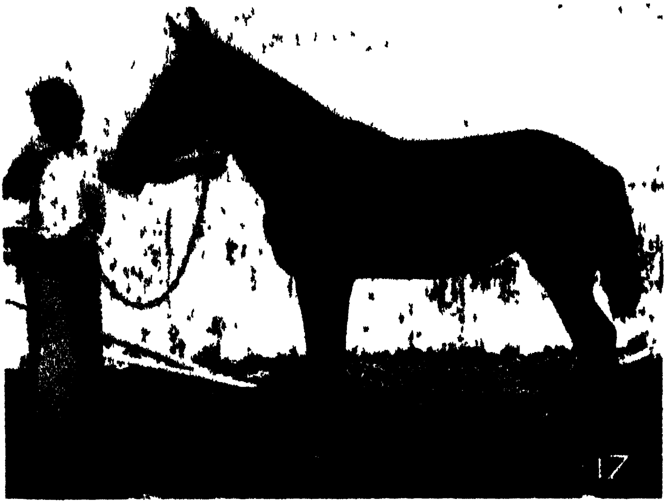


Fig. 17. Horse 20723.

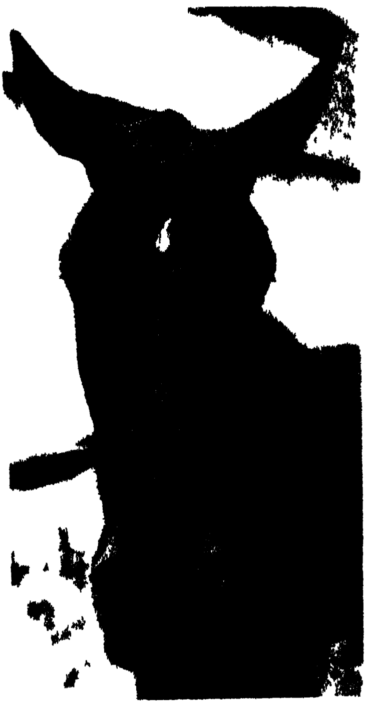


Fig. 18. Face of Horse 20722.

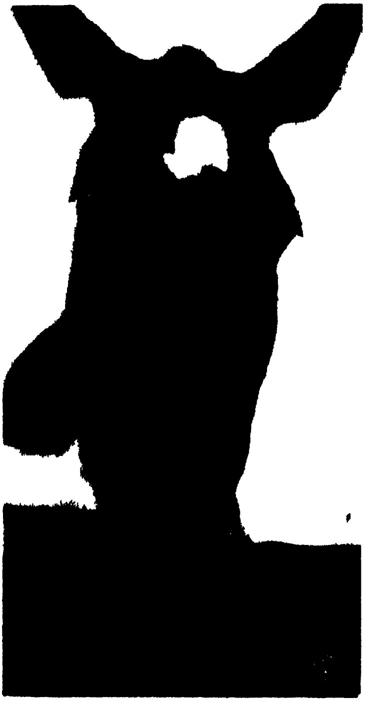
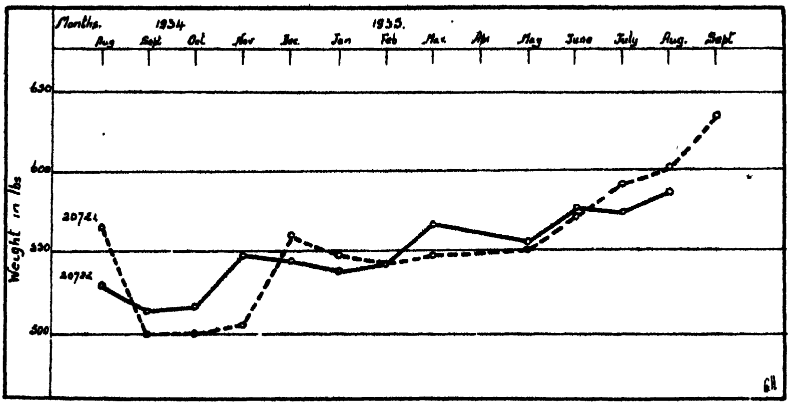


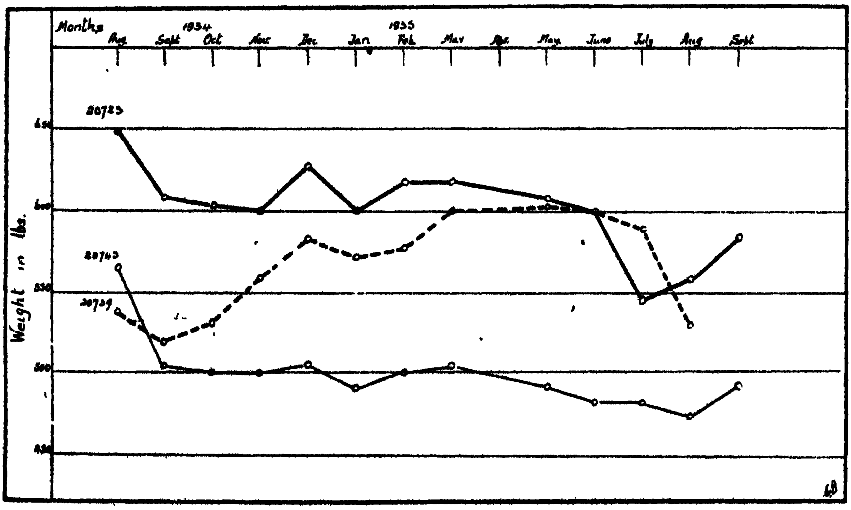
Fig. 19. Face of Horse 20723.

APPENDIX 2.

Weight Curves: Horses 20721 *and* 20722.



Weight Curves: Horses 20723, 20739 *and* 20745



APPENDIX 3.

Inorganic Phosphorus in mgm./100 c.c. blood.

Number.	Months: 1935.					Months: 1936.								
	5.	6.	9.	10.	11.	1.	3.	4.	5.	6.	7.	8.	9.	10.
20723*	2·9	4·9	3·6	3·9	4·0	3·4	3·5	3·7	3·6	3·7	3·3	4·0	4·0	3·9
20739	3·3	4·4	3·4	die	d.									
20721	1·8	2·9	3·3	die	d.									
20722	1·9	4·0	3·6	3·6	2·8	5·1	2·9	3·7	3·8	3·4	3·7	3·5	3·9	3·8
20745	3·1	3·4	3·9	4·0	5·1	4·6	8·5	2·6	5·6	5·0	4·9	5·8	5·5	5·9

* 20723 received a $CaCo_3$ supplement in 1936.

I.P. Serum.

Number.	5.	6.	9.	10.	11.	1.	3.	4.	5.	6.	7.	8.	9.	10.
20723	5·2	5·2	5·2	4·7	5·0	4·8	4·9	5·2	5·0	4·0	5·1	5·3	5·0	—
20739	6·1	5·1	4·9	die	d.									
20721	4·8	4·1	4·0	die	d.									
20722	4·1	3·6	5·1	4·5	4·2	—	3·9	5·0	5·1	4·5	4·6	4·5	5·2	—
20745	5·3	4·7	4·7	4·4	6·8	—	10·5	8·2	6·4	6·3	5·8	6·9	7·2	—

Phosphatase.

Number.	5.	6.	9.	10.	11.	1.	3.	4.	5.	6.	7.	8.	9.	10.
20723	10·9	12·4	8·1	9·1	12·2	—	13·2	10·1	13·0	11·5	11·0	9·0	13·3	—
20739	9·5	11·0	11·6	die	d.									
20721	7·1	8·4	7·3	die	d.									
20722	11·8	10·1	10·0	10·0	12·8	—	11·5	10·1	15·6	11·8	13·0	11·1	13·4	—
20745	6·5	6·5	6·7	7·7	9·4	—	8·5	8·1	10·8	8·0	10·8	11·4	9·8	—

Calcium.

Number.	5.	6.	9.	10.	11.	1.	3.	4.	5.	6.	7.	8.	9.	10.
20723	—	—	10·9	10·1	10·8	—	11·1	8·3	7·9	10·1	9·6	9·4	9·1	—
20739	—	—	11·1	die	d.									
20721	—	—	9·6	die	d.									
20722	—	—	9·4	10·6	10·8	—	10·1	9·0	8·9	10·1	9·7	10·1	9·5	—
20745	—	—	11·2	10·7	8·8	—	9·0	8·4	8·4	8·3	7·2	9·4	6·9	—

Haemoglobin.

Number.	5.	6.	9.	10.	11.	1.	3.	4.	5.	6.	7.	8.	9.	10.
20723	—	—	12·1	12·4	11·3	—	—	—	—	—	—	—	—	—
20739	—	—	11·7	die	d.									
20721	—	—	11·9	die	d.									
20722	—	—	11·5	12·1	12·0	—	—	—	—	—	—	—	—	—
20745	—	—	11·8	11·7	12·0	—	—	—	—	—	—	—	—	—

Appendix 4.

RADIOGRAPHS OF METACARPAL BONES OF HORSES 20722, 20723, 20721, 20739 AND 20745.

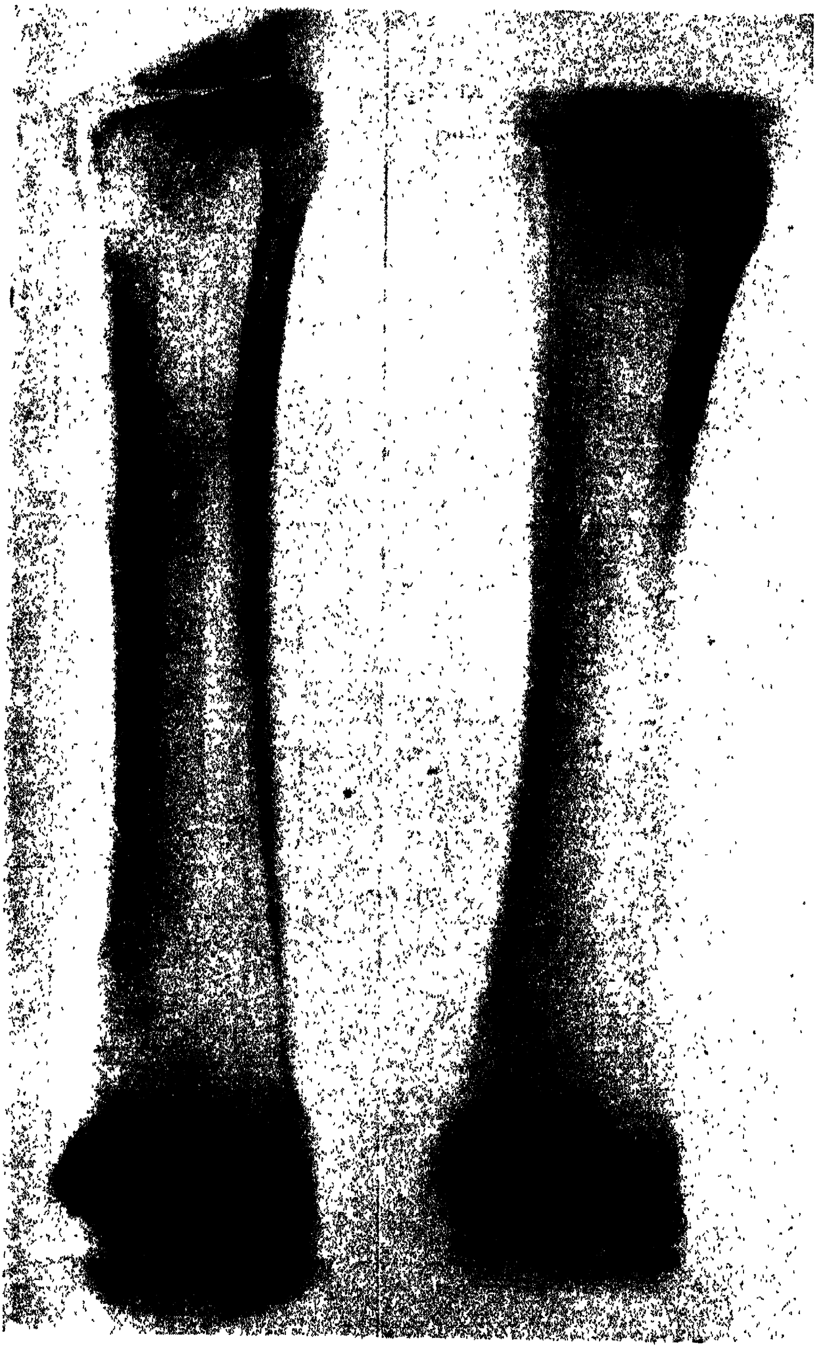


Fig. 20. Horse 20722.

Fig. 21. Horse 20723.

Fig. 22. Horse 20721.

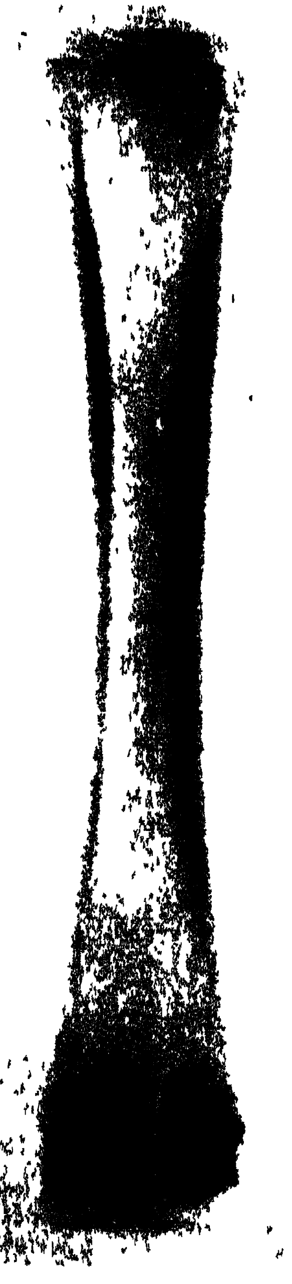


Fig. 23. Horse 20739.

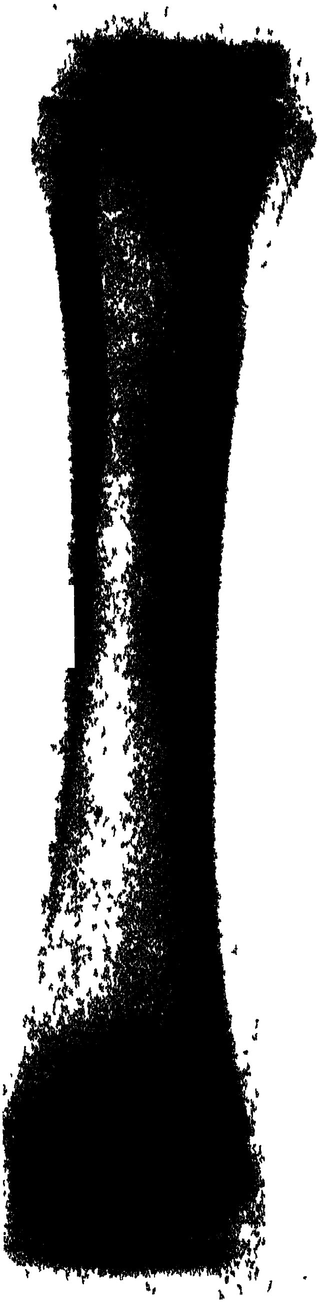


Fig. 24. Horse 20745.

Section VI.

Chemical Pathology.

VAN NIEKERK, P. LE R. Veterinary biochemical studies I. A rapid method for the determination of copper in biological material ... 623

Onderstepoort Journal of Veterinary Science and Animal Industry, Volume 9, Number 2, October, 1937.

Printed in the Union of South Africa by the Government Printer, Pretoria.

Veterinary Biochemical Studies.

I.—A Rapid Method for the Determination of Copper in Biological Material.

By P. LE ROUX VAN NIEKERK, Section of Chemical Pathology, Onderstepoort.

Of the micro-methods which have been proposed for the determination of copper, the method of Biazzo (1926) as modified by Elvehjem and Lindow (1929) has been found to be the most practicable. Until recently McFarlane, (1932), the accuracy of this method has never been questioned. At this laboratory the method was found to give fairly satisfactory results when applied to biological material. In the cases of spleen and blood where the presence of excess iron, due to the large aliquots necessary, interfered with the colour development, the copper had to be separated from the iron at a carefully adjusted pH.

The discovery by Callan and Henderson (1929) that when an aqueous solution of sodium-diethyldithiocarbamate is added to a solution containing copper, a golden brown colour is developed with the formation of a normal copper salt of diethyldithiocarbamic acid. This colour reaction forms the basis for the method discussed here and has lately found much favour in the colorimetric determination of copper.

MacFarlane (1932) extracted the colour quantitatively with amyl alcohol, thereby intensifying the colour and increasing the sensitivity of the method.

It was found necessary, on account of the great number of analyses to be done here, that a rapid and accurate method was essential, and for this purpose the methods of MacFarlane (1932) and Tompsett (1934) were modified and applied as described here.

A large number of copper determinations was done by this method in conjunction with certain copper experiments at present in progress at Onderstepoort. These determinations were done on both normal and pathological post-mortem material as well as on several hundred grass and shrub samples.

These results will be incorporated in later publications from this Institute, in a study on the rôle of copper in certain stock diseases.

REAGENTS.

1. *Copper Standard.*—Dissolve one gram of purest electrolytic copper in sufficient concentrated nitric acid, dilute and make up to 100 c.c. with distilled water. Dilute 10 c.c. of this solution to one litre which gives a stock solution containing ·1 mgm. Cu per c.c. Dilute this again to obtain a working standard of ·01 mgm. Cu per c.c.

2. *Magnesium Oxidizing Mixture.*—Shake up a 20 per cent. aqueous solution of magnesium nitrate with magnesium carbonate until saturation, filter and bottle.

3. Make a ·5 per cent. aqueous solution of sodium-diethyldithiocarbamate, filter if necessary, and keep in a dark coloured flask.

4. Saturated solution of sodium citrate.

5. 4 per cent. solution of sodium pyrophosphate.

6. 20 per cent. solution of ammonium hydroxide.

It is essential to use only the purest chemicals and in all events do blank tests upon them.

All porcelain or silica dishes should be periodically cleaned according to Elvehjem and Lindow (1929) as follows:—

Put one gram of sodium acetate into each basin and dissolve in sufficient alcohol, evaporate and ignite. The contents are then extracted with hydrochloric acid 1:1 for several days.

MATERIAL.

Use 10 grams of liver and 20 grams of spleen, kidney, lung and 20 c.c. blood, respectively, for each determination. In the case of faeces and food use 5-20 grams according to the amount of copper expected to be present.

METHOD.

In all cases weigh the material into 50 c.c. silica dishes and add 5 c.c. of the magnesium oxidizing mixture. If this is omitted it is found that on ignition even at fairly low temperatures a certain amount of slagging of the copper with the silica of the dishes takes place, consequently recovery of copper in the determination is low as can be seen from the following data:—

In this case liver was finely minced and well mixed. Known amounts of copper were added to the weighed out quantities of liver prior to ignition. The copper content of the liver was determined with the addition of magnesium mixture and the average result of five determinations taken. In the cases where no magnesium mixture was added the slagging was visible to the naked eye and results were low.

The average copper content of the liver was found to be 0·326 mgm. See Table I.

TABLE I.

No.	C.c. of magnesium mixture added.	Mgm. copper in liver.	Mgm. copper added.	Mgm. copper recovered.	Mgm. copper not accounted for.
1	0	0·326	0·0	0·164	− 0·162
2	0	0·326	0·5	0·525	− 0·301
3	0	0·326	1·0	1·020	− 0·306
4	0	0·326	2·0	2·084	− 0·242
5	0	0·326	3·0	3·150	− 0·176
6	5	0·326	0·1	0·448	+ 0·022
7	5	0·326	0·5	0·832	+ 0·006
8	5	0·326	1·5	1·766	− 0·060
9	5	0·326	2·0	2·226	− 0·100
10	5	0·326	3·0	3·256	− 0·070

The time taken for the complete ignition of the samples without the addition of magnesium nitrate mixture was approximately twice that of the last five samples in Table I. It is thus clear that the complete ignition of biological material, without an adequate oxidizing agent is very time-consuming and unsatisfactory. The effectiveness of this oxidizing agent is especially noticeable in the cases of livers of stock that have been dosed with copper salts or where copper has been added to the sample. In such cases the green copper salt can be seen to be concentrated largely at one spot lying on top of the magnesium salt, without making contact with the surface of the basin.

The ignition can be carried out at fairly high temperatures, without the loss of copper either due to slagging or conversion into insoluble compounds. In fact, an added advantage of a fairly high temperature is that a large per cent. of iron is rendered insoluble.

After incineration is complete, extract the contents of the dishes for about 30 minutes with 5 c.c. concentrated nitric acid. This can be accomplished without heating on account of the readily soluble form in which the copper is present. Dilute the contents with 5 c.c. water, filter carefully through Whatman No. 40 paper into 50 c.c. volumetric flasks. Wash thoroughly with distilled water up to volume.

Take 0·5-5 c.c. aliquots of liver extract, 5-10 c.c. of kidney and 20 c.c. of lung, spleen and blood extracts, respectively.

Pipette these aliquots into 50 c.c. test tubes and add water till about 20 c.c. volume. To these add 4 c.c. of 4 per cent. sodium pyrophosphate solution. According to McFarlane (1929) this is added in order to suppress the ionisation of the iron compounds. It was, however, found that it also improves the separation of the iso-amyl alcohol from the solution containing the copper.

For each 5 c.c. aliquot used now add 2·5 c.c. of saturated sodium citrate solution, e.g. 10 c.c. per 20 c.c. spleen extract. This prevents the precipitation of magnesium and calcium salts, which would absorb some of the colour formed later on in the procedure and consequently too low values would be obtained as can be seen from Table II.

TABLE II.

C.c. sodium citrate added.	Mgm. copper added.	Mgm. copper recovered.	Mgm. copper not accounted for.
0	0·08	0·065	− 0·015
0	0·16	0·128	− 0·032
0	0·16	0·133	− 0·027
5	0·07	0·070	0·0
5	0·08	0·078	− 0·002
5	0·16	0·163	+ 0·003

Add 20 per cent. ammonium hydroxide until alkaline to litmus. Add exactly 10 c.c. of iso-amyl alcohol and ·5 c.c. of the ·5 per cent. solution of sodium-diethyldithiocarbamate reagent. Swirl the tube and close with the hand or stopper to shake the contents vigorously for half a minute. Treat several standards containing ·01, ·02, ·03, and ·05 mgm. Cu respectively exactly in the same manner. Pipette about 8 c.c. of the amyl alcohol into 10 c.c. centrifuging tubes and centrifuge out any water present at approximately 3,000 r.p.m. for 5-10 minutes. Decant the supernatant alcoholic layer into colorimeter cups and read against the standard most closely corresponding in colour. The use of ·5 c.c. of a ·5 per cent. solution of the carbamate reagent differs from the recommendation of McFarlane (1932) who uses ·5 c.c. of 2 per cent. strength. It was, however, found that ·5 c.c. of ·5 per cent. strength is sufficient to precipitate 0·3 mgm. copper, i.e. approximately fifteen times the amount of copper usually taken for a determination.

The main object for this change is that when more of the reagent is used a precipitation of iron tends to occur which does not happen when the lesser amount is employed. This interference of iron may be eliminated, as McFarlane stated, by the addition of one drop of a 40 per cent. sodium hydroxide solution and heating for fifteen minutes at 80° C. in water. It was found in cases of organic extracts containing relatively much iron, as well as in blank determinations to which iron had been added, that this boiling with the addition of alkali could be omitted without giving too high values as a result of the coloured iron precipitate formed when more of the reagent is used. This can be seen from Tables III and IIIA.

TABLE III.

Blank Determinations.

Mgm. copper added.	Mgm. Fe added.	Treatment.	Mgm. copper recovered.	Percentage copper recovered.	Remarks.
·02	0·0	Heated at 80° C. + alkali + ½-c.c. of 2 per cent. reagent	·020	100	
·02	0·0	,, ,,	·019	95	
·02	1·0	,, ,,	·018	90	
·02	2·0	,, ,,	·018	90	
·02	3·0	,, ,,	·019	95	
·02	4·0	,, ,,	·020	100	
·02	5·0	,, ,,	·020	100	
·02	0·0	Used ½-c.c. of 2 per cent. reagent without boiling or alkali	·020	100	
·02	0·0	,, ,,	·021	105	
·02	1·0	,, ,,	·020	100	
·02	2·0	,, ,,	·020	100	
·02	3·0	,, ,,	·021	105	Iron did not interfere.
·02	4·0	,, ,,	·021	105	
·02	5·0	,, ,,	·024	120	Iron definitely interfered.
·02	10·0	,, ,,	·025	125	
·02	1·0	Used ½-c.c. of ·5 per cent. reagent without boiling or alkali	·020	100	
·02	2·0	,, ,,	·020	100	
·02	3·0	,, ,,	·019	95	
·02	4·0	,, ,,	·020	100	
·02	5·0	,, ,,	·019	95	
·02	10·0	,, ,,	·023	115	Iron interfered.

TABLE IIIA.

Determination on Spleen Extracts.

Amount.	Treatment.	Mgm. copper found.
20 c.c.	Heated at 80° C., + alkali + ½-c.c. of 2 per cent. reagent	0·023
20 c.c.	,, ,, ,, ,,	0·025
20 c.c.	,, ,, ,, ,,	0·023
20 c.c.	Without heating or adding alkali, + ½-c.c. of ·5 per cent. reagent	0·025
20 c.c.	,, ,, ,, ,,	0·024
20 c.c.	,, ,, ,, ,,	0·022
20 c.c.	,, ,, ,, ,,	0·022

PLANT MATERIAL.

Although these analyses were made on filtrates obtained by the method of extraction of Louw (1934), it was nevertheless found that the plant material could be treated and extracted in the same manner as the other biological material as described herein. The results obtained by these two different ways of extraction compared favourably.

SUMMARY.

1. An accurate and rapid method, which is a modification of the methods of McFarlane (1932) and Tompsett (1934) for the determination of copper, has been presented.

2. An oxidizing agent in the form of magnesium nitrate and carbonate is used to accelerate the ignition and render the copper easily soluble.

3. The quantity of sodium di-ethyldithiocarbamate is decreased to eliminate the interference of iron compounds usually found in organic material.

4. The addition of sodium citrate solution to render the calcium and magnesium salts soluble in the ammoniacal solution is discussed.

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In conclusion I wish to place on record my appreciation of and indebtedness to Dr. P. J. du Toit, Director of Veterinary Services, for placing all the required facilities at my disposal and permitting the publication in the Onderstepoort Journal.

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Section VII.

Animal Husbandry.

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Animal Husbandry of the Hottentots.

By Dr. H. EPSTEIN.

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1. THE COUNTRY OF THE HOTTENTOTS.

DURING the early days of the Dutch settlement at the Cape, the Hottentots occupied most of the western half of South Africa. They were thinly scattered over this vast stretch of land, in small loosely-organised groups, each with its own individual name. The Inqua, Damaqua and Gonaqua constituted the most eastern tribes along the south coast of the Cape, the Gonaqua extending as far east as the Great Fish River; whilst the Great Namaqua were the northernmost group, living north of the Orange River in the southern parts of what is now South West Africa. But scattered hordes of Great Namaqua extended even into the north of that country, the ||Khau-|Goan and ‡Aunin (Topnaars) wandering as far north as the Etosha Pan and the Kaokoveld, south of the Kunene River.

Originally the Hottentots do not appear to have camped anywhere far from the coast belt. The !Kora, for instance, who at the beginning of the last century were found round the junction of the

Vaal and the Harts, were originally resident in the Cape Peninsula, and had only been forced into the interior of the country through their continual strife with the Europeans, and later through their quarrels with the Bantu. The same applies to such scattered groups, remnants of different tribes, as the Amraal Hottentots, round Gobabis, and the !Khara-GeiKhoin, south-east of Rietfontein S., British Bechuanaland, who left the coastlands only when the Hottentot peoples carried out a general movement away from the European settlements. (Schapera, 1930.)

The climate of the original habitat of the Hottentots is determined by the physical conformation of the land and by its proximity to the Equator. A narrow fringe round the coast has an elevation of less than 1,500 feet above the sea, whilst most of the interior consists of a high plateau rising gradually around its borders to an elevation of over 3,000 feet. On the west, the escarpment is separated from the Atlantic by fairly uniform slopes, whilst in the south the Great Karroo and folded mountain ranges intervene. The high plateau intercepts the rainbearing winds from the Indian Ocean, so that the climate along the west coast is extremely dry; but along the south coast and the south-west the rainfall is heavier. Over the south-western Karroo the mean annual rainfall is 10 inches, being reduced towards the north-west of South West Africa to less than 3 inches. In the Cape Peninsula, on the other hand, the rainfall reaches an average of 40 inches per annum.

Along the west coast from the Olifants River to the Kunene stretches the barren desert country of the Namib, varying in width from 18 miles in the north to about 85 miles in the south. The greater part of the Namib is covered with drifting sand dunes, the monotonous character of the land being varied in places by more broken country. This stretch of coastland is dominated by the cold antarctic Benguella current whose power is rarely countered by very hot winds from the east. The sporadic rain falls during the summer from October to April.

South of the Orange River the Namib gradually changes into the highlands of Little Namaqualand where the mean annual rainfall is slightly higher than in the desert, reaching as much as 15 inches. The country south of the Olifants River is characterised by folded mountain ranges, separated by wide valleys, which extend over the whole southern portion of Cape Province. The total annual rainfall of this region reaches an average of 30 inches. The rain falls during summer and winter, but its seasonal distribution varies greatly according to locality. The Cape Peninsula itself is the best-watered area of what was formerly Hottentot land. The bulk of the rain falls during the winter, April to September, the summer being the dry season. East of the Cape Peninsula, the rainfall decreases again to between 15 and 20 inches.

Thus the land of the Hottentots is for the most part arid and semi-arid country. Its range extends through three distinct rainfall areas, the area of summer rain and winter drought, a second where rain may occur any time of the year, broken by irregular periods of drought, and finally, the winter rain area.

The rivers and water courses correspond with the great diversity of the climatic and physical conditions of Hottentot land. In the far north-west, between the highland region of the Kaokoveld and the Etosha Pan, the sandy plain is intersected by a number of shallow water courses which are filled by the overflow of the Kunene River and drain to the Etosha Pan. The hilly country south of the Kaokoveld, i.e. Northern Damaraland, is devoid of defined waterways, sink holes and subterranean caverns and springs. Along the coast, however, the Namib, at least in its northern portion, is distinguished by the presence of water courses flowing through open channels to the sea. In a southerly direction, where the Namib and the western outskirts of the Kalahari desert pass into Great Namaqualand, the Anas Mountains form the watershed of South West Africa. This part of Hottentot land is drained by the Kondip and Great Fish Rivers which carry their waters to the Orange. The Orange and Berg Rivers are practically the only streams of Hottentot land retaining their surface water all the year round. Almost all the others are dry sandy river courses which carry water merely for a short period after rain; and only in very wet seasons do they keep up their flow for any length of distance. The rest of the time they have water below their beds in places far between, or in stagnant pools, or occasionally from a spring in the otherwise dry river course. Away from the river beds springs are very few and water hard to find, as the hollows in the rocks and the vleis and depressions hold it only for a short time after heavy rains.

South of the Orange River, in the highlands of Little Namaqualand and the arid plain of Bushmanland, enclosed by the great bend of the Orange River, there is practically no surface water at all, the river beds being dry throughout the greater part of the year. But in the Kamiesberg, south of Little Namaqualand, a few short perennial streams are met with.

The southern portion of Hottentot land is formed by the Central or Great Karroo of the Cape Province which rises to 3,000 feet above the sea, and extends from the Olifants River in the West to the Sundays River in the east. Its river beds are dry, except during the short periods of summer rainfall when the waters sweep down to the sea. In the dry season they may turn into a series of pools and gullies, and in times of severe drought dry up completely. This applies even to such important rivers as the Gouritz, Gamtoos and Sundays, which have their sources in the summer rain region behind the coastal ranges. Only a few smaller streamlets situated in the limited area of moderately high winter rainfall rarely dry up. But the Great Karroo, north-east of this small winter rain area, in the days of the Hottentots when there were no wells or boreholes, was a desert proper.

The amount and incidence of the rainfall and scarcity of perennial surface water explain very largely the existing flora in the territory of the Hottentots.

The Kaokoveld and Ovamboland consist of elevated plains thickly covered with red sand and calcareous deposits. The rainfall, though very small, permits a fair amount of vegetation, mainly grasses, shrubs and trees. The plains are covered with tough Bushman grass (*Aristida brevifolia*) or dense thornbush. *Aloe dichotoma* and *Euphorbia dinteri* as well as various leguminous and bulbous plants are characteristic of the vegetation of these regions. Over the arid tract of the Namib a very scanty xerophytic vegetation is encountered, whilst considerable areas are practically devoid of any growth except the remarkable tsama melon (*Citrullus vulgaris*) which, owing to its high water content, is the most important plant throughout the arid parts of Hottentot territory during the height of the dry season. (Schapera, 1930.) The sparse covering of cacti and stunted, dwarfed bushes, such as the milk bush (*Euphorbia sp.*) is sustained in the rainless Namib chiefly by the wet and heavy sea fog.

In Damaraland and Great Namaqualand the vegetation is richer than in the Namib. Northern Damaraland is a tract of extensive grass plains, the low-lying country often extremely fertile. The flora of this region includes the inflammable candle bush (*Sarcocaulon Burmanni*), the unique *Welwitschia mirabilis*, and, above all, the leafless cucurbitaceous !naras (*Acanthosicyos horrida Welw.*), an edible melon-like fruit. In the river beds the leaves and pods of various Acacias, especially the Kameeldoorn (*Acacia giraffae*) and the Ana (*Acacia albida*), provide a rich verdure and staple feed for cattle and sheep in these parts. The northern districts of Great Namaqualand form a park-like savanna with large thorn trees and well timbered stretches along the river beds; Kameeldoorn and Kokerboom (*Aloe dichotoma*) are most prominent in this region. In the south of Great Namaqualand, semi-desert conditions prevail, the arid plateaux supporting a poor growth of grass and shrubs, the latter mostly being of the Grevia species. During seasons with a heavy rainfall nutritious grasses crop up everywhere.

The arid and semi-arid tracts of Bushman- and Little Namaqualand produce a similar vegetation to the country adjoining the Orange in the north. The general aspect of the flora is that of widely separated xerophytic shrubs and bushes interspersed with various succulent plants. Grasses occur rarely, always growing in tufts.

In the Upper Karroo, south and east of Bushmanland, there is a considerable diversity of vegetation owing to the great range in rainfall and altitude. The north-western parts have a proper desert flora, while the more favoured districts show the characteristic vegetation of a semi-desert. A variety of dwarfed and stunted shrubs and bushes, mostly of the family Compositae, provide a sufficient sustenance for large numbers of stock even throughout the driest seasons. Grasses shoot up during the rainy season, but these do not form a typical feature of Upper Karroo vegetation. Thorn bushes and succulent, bulbous and tuberous plants are the characteristic flora of this region, while Acacias border the intermittent river courses.

South of the Olifants River, the eastern part of the Goudini valley adjoins the Southern Karroo which is almost entirely destitute of vegetation, whilst its southern part passes into the Cape Peninsula abundantly supplied with its winter rainfall. The characteristic Cape flora is dominated by a large variety of evergreen shrubs and numerous bulbous, tuberous and similar plants possessing subterranean storage organs. The sandy plains of Cape Peninsula, its marshy hollows, river banks and mountain sides, all have their own peculiar flora. Patches of forest are found on the seaward sides of the mountains, whilst in the plains the flora is chiefly composed of Proteaceae, such as the silver tree (*Leucadendron argenteum*), the sugar bush (*Protea mellifera*) and other evergreen shrubs, Restionaceae, a family of grass-like plants, Ericaceae,, the typical plants of the heath, and various grasses proper.

In the Great Karroo vegetation is very scanty, and during the dry season the soil entirely devoid of verdure. Drought-resisting thorn bushes, succulent, bulbous and tuberous plants compose the typical Karroo flora. Along the river courses and on the eastern mountain slopes trees occur in sporadic patches; during exceptionally wet seasons even grasses occasionally crop up.

These are the climatic and floral conditions of the country of the Hottentots, poorer than anywhere else throughout the southern parts of Africa. They have set their stamp on the Hottentots' mode of life and on their pastoral industry.(¹)

2. THE DOMESTIC ANIMALS OF THE HOTTENTOTS.

When Europeans first set foot upon South Africa, they found the country inhabited by Hottentot tribes in possession of large herds of cattle and flocks of sheep. Van Riebeeck, the founder of the Dutch settlement at the Cape, describes the herds of the Hottentots in the surroundings of Table Bay as being as numerous as blades of grass in a field. Akembie, the chief of the Namaqua Hottentots, is said to have alone possessed in 1661, 4,000 head of cattle and 3,000 sheep (Stow, 1905). Besides cattle and sheep, the only other domestic animal possessed by the Hottentots was the dog. From van Riebeeck's statement it is evident that none of the Cape Hottentots bred goats, and this applied to other Hottentot tribes as well. The goat was never used in their ceremonial meals, if it could possibly be avoided. This in all probability, suggests Mrs. Hoernlé (Hoernlé 1918), is due to the fact that it was not one of the original domestic animals of the Hottentots. They acquired goats some time later from the Bantu with whom they came in contact shortly after the European occupation of the Cape. The Naman, for instance, obtained their goats by barter from the BaThlaping, a Bantu tribe whom they called Biriqua, i.e. goat people (Dapper, Ten Rhyne and De Grevenbroek, 1933), and this is also the Hottentots' usual name for the BeChwana (Schapera, 1930).

The Hottentot cattle were gaunt, bony creatures. According to Kolben they were bigger and stronger than European breeds, a Hottentot ox weighing about 5-600 lb. (Kolben, 1719). The head was long and narrow, especially the nasal part, but with a fairly broad forehead; the body moderately deep and broad, and the legs strong, clean and well placed. The tail was long and thin, with a well developed switch, and the horns long and slender, their direction

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(¹) This chapter is compiled mainly from the following sources: I. Schapera, The Khoisan Peoples of South Africa, 1930, Chap. I.; H. Epstein, The Red Afrikander Cattle, Chap. I (unpublished); S. Passarge, Südafrika, 1908; The Oxford Survey of the British Empire, 1914, vol. on Africa.

being mainly outward and with a slight twist. This peculiar direction of the horns may have led Stow (1905) to conclude erroneously that the Namaqua had a practice of training the horns of their oxen artificially, confining their shape to a spiral line similar to that in the Koodoo. But this observation may also be based on an entirely different phenomenon. Sparrman (1789) writes that in the Zuurevelden the cattle are sometimes given to gnaw each others' horns when shut up in their kraals at night, through which their horns take on a carved appearance; " a circumstance which ", says Sparrman, " ought therefore by no means to be ascribed, as it has been, solely to the ingenuity and manual operation of the herdsmen". (See fig. 1.)

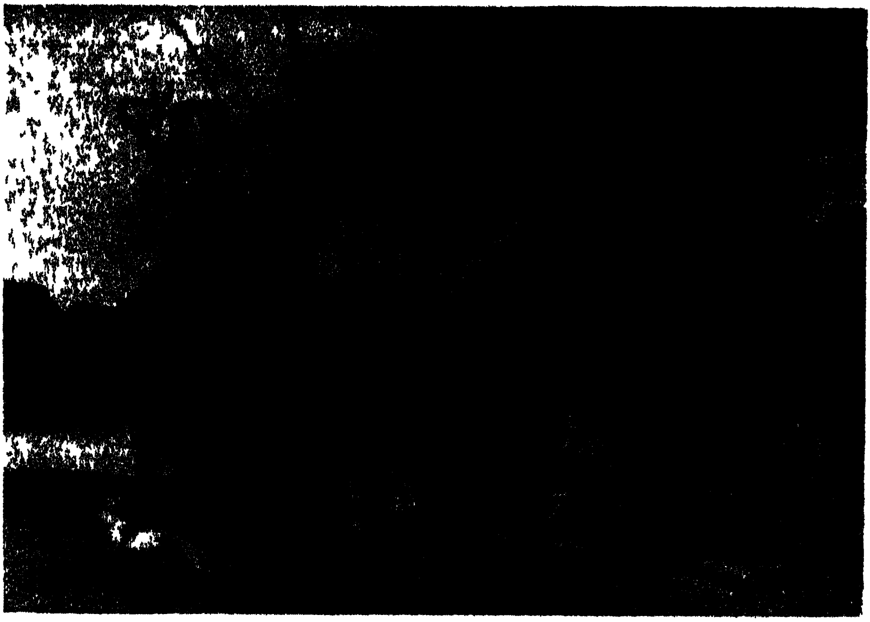

Fig. 1 —Korah Hottentots preparing to move. After Samuel Daniell.

The hump of the Hottentot cattle does not seem to have been very strongly developed. On Daniell's picture the load obstructs, judging the direction of the top line of the ox and the development of the hump, but the position of the front pack indicates a considerable slope down of the back behind the withers.

" The majority of authors ", says Kolben (1719), " who have described Hottentot cows and oxen, state that these are distinguished by large humps. But I can assure my readers that, although I have seen the herds of the Dutch settlers and of many Hottentots, I have never met with a humped beast. The above statement is therefore either untrue, or the yoke pressing upon the neck of the ox causes the withers to appear raised to a hump. Yet it is certain that by nature their cattle are not humped though they are larger and stronger than European breeds ".

This statement is difficult to accept as it stands. Kolben himself says that most of the other writers describe the Hottentot cattle as humped. On Daniell's and others' paintings of Dutch settlements, dating from the end of the eighteenth and the beginning of the nineteenth century, a large proportion of the oxen are depicted with humps. Yet Kolben (1719) himself states that " as the Hottentots know, the cattle of the Europeans originate and descend from the Hottentot's own stock ".

But the fact that Kolben does not credit Hottentot oxen with humps is not surprising considering that the Hottentots lived, at the time of the European penetration into southern Africa, in one of the driest and poorest parts of the subcontinent. With all zebu cattle the size of the hump is easily influenced by the feeding. For instance, it is frequently observed in zoological gardens that zebu calves born there develop tremendous humps, whereas in their imported parents the hump is hardly noticeable (Epstein, 1937). In poorly fed specimens of the red Afrikander breed, which is descended as pointed out in another place from cattle of Hottentots (Epstein, 1933), the hump is even nowadays practically unnoticeable. Yet the hump of the Afrikander is one of the chief characteristics of this breed; and it is evident that this feature could not have been developed by Cape settlers, had its genetic factor not already been present in the original Hottentot stock.

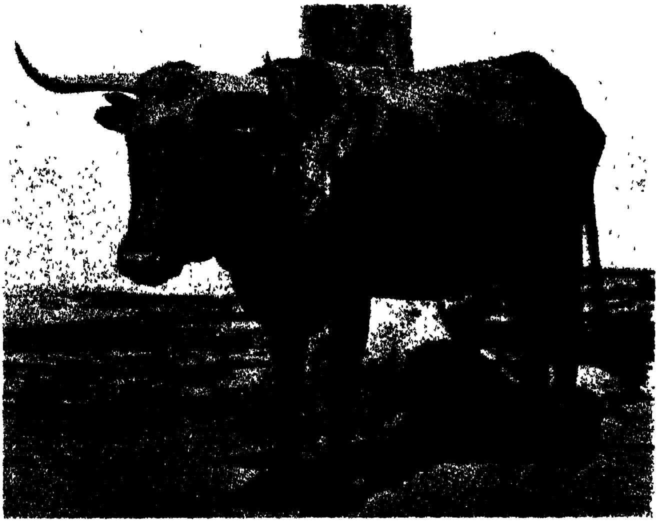

Fig. 2.—Hottentot cow. After Schultze.

The accompanying photo of a cow of Hottentot type reproduced by Schultze, shows an animal which an untrained observer would describe as humpless. But it is evident to a trained eye that this cow would show a proper hump when well fed, and would certainly be capable, with a bull of similar conformation, of producing a calf that would develop a very prominent hump if properly reared. (See fig. 2.)

The principal colour of the Hottentot cattle was red through every shade from darkest to light. The ox represented on Daniell's painting is dark red, and the cow reproduced by Schultze seems to have been the same shade also. Black Hottentot cattle too were fairly common, and there were many red animals which had a white top and underline, or their red colour broken by white markings on almost any part of the body including the switch, and especially on the belly, dewlap and flanks. Roan was practically unknown among Hottentot cattle. But a red background with small white spots along the belly, dewlap, brisket, flanks and ribs was not rare. Brown and yellow stock occurred fairly frequently. In very rare instances Hottentot cattle were of a creamy white, even of an almost snow-white colour.

Hottentot sheep are relatively large in body, although not standing particularly high. Their heads are fairly big and long, with a convex, flat or occasionally even concave profile, and a slightly convex or straight chaffron. The muzzle is narrow and rounded. The eyes are unusually small, the ears fairly long and pendent, not too broad and narrowing slightly towards the tips. The ewes are mostly hornless, but the rams carry horns which are either crescent-shaped or elongated into a horizontal spiral. Their horns are fairly large tapering off rapidly towards the tips and displaying a great number of distinct transverse wrinkles. (See fig. 3.)

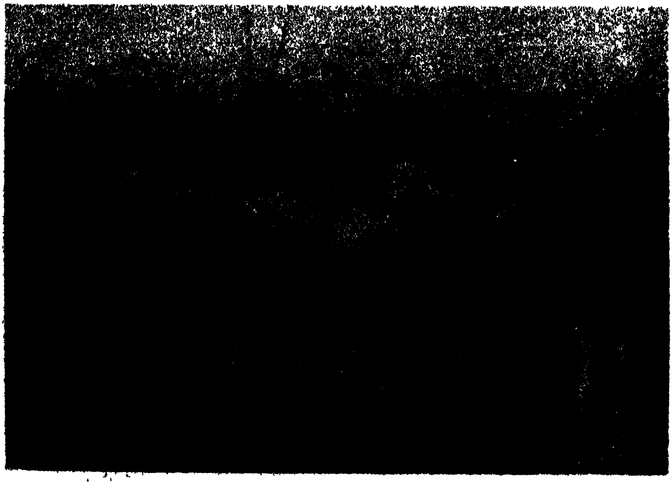

Fig. 3.—Hottentot sheep. After Antonius.

Usually the tail reaches to the hocks where it terminates abruptly, but in some specimens its cylindrical tip is so long as to sweep the ground. In the hairless under-surface of its fat-laden portion, and in the separation of the bare from the hairy areas by a pair of longitudinal grooves, the tail of the Hottentot sheep resembles that of other fat-tailed breeds. In most cases, however, there is a smaller accumulation of fat than in the majority of African and Asiatic fat-tailed animals. In some instances the fat-tail is egg-shaped not even reaching the hocks. In short, the modifications of tail structure displayed by the Hottentot sheep are numerous, hardly two animals being exactly alike in this feature. The tail generally weighs about three to four kg. (Theal, 1907). (See figs. 4 and 5.)

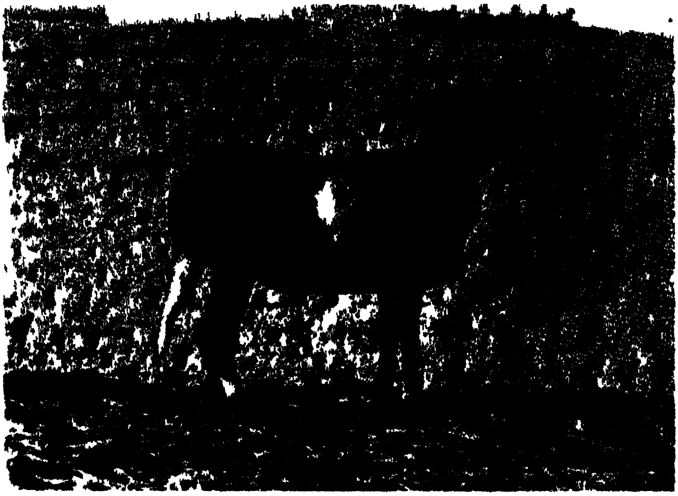

Fig 4.—Nama ram After Schlettwein

Fig. 5.—Nama ewe. After Schultze.

The majority of Hottentot sheep are covered with hair instead of wool, the hair being short, straight and coarse. Others have a thick and curling woolly fleece, but in some specimens the fleece is long, coarse and shaggy. On the face, belly and legs the hair is short and dense. The rams usually display a more or less distinct throat mane.

Their colour varies from uniform dirty white to rusty or dark brown, black or silver grey, but piebald or skewbald specimens are not unknown.

The dogs of the Hottentots were ugly creatures, their bodies being shaped like jackals', and their hair coarse and bristling forward on the spine (Theal, 1907). They varied in size and colour. Schinz (1891) describes them as of medium size, with short hair, long snouts, and drooping ears; Von François (1896) as coarse and ugly, and varying in size and colour; whilst others refer to them as lean, hungry looking mongrels, half starved and savage-tempered.

3. Descent and Origin of the Hottentot Cattle.

The amount of research that has been hitherto devoted to solve the question of the racial origin of the Hottentots' domestic animals is negligible. For this reason practically all opinions advanced on this point have proved erroneous. Johnston (1908), for instance, in mentioning the resemblance of Hottentot and Bantu roots of the terms for cattle, sheep and goats, argues that the goat first, then the ox and the sheep were brought to the Hottentots from the north by Bantu or Nilotic negroes. In a previous publication on the West African Shorthorn the author (1934) has already commented on the superficiality of Johnston's hypotheses on the origin of African domestic animals. In the case of the domestic breeds of the Hottentots it is particularly dangerous to come to conclusions solely from philological resemblances of names, without producing any supporting evidence. That such conclusions are often liable to error is shown by the following instance: The Hottentots use the term "hâs" for mare, from which the typically Hottentot masculine "hâb" for stallion has been formed. If we were to view this linguistic phenomenon independently of any other considerations, we would arrive at the wrong conclusion that the Hottentots acquired their first horses from English speaking people. This however is not the case. The first horses were imported into South Africa by the Dutch settlers who used the Afrikaans name "perd".

As to Johnston's theory of the Bantu origin of the Hottentot's domesticated animals, it is well known that within at least historical times the Hottentots possessed sheep and cattle and an old pastoral tradition long before they came into contact with the Bantu. It is true they obtained goats from the Bantu after the European occupation of the Cape. But even in this case Johnston errs when arguing from philological resemblances that the Hottentots obviously acquired their goats prior to their cattle and sheep.

Whether the Hottentots obtained their domestic animals from the Bantu in East Africa is, as Schapera (1930) submits, a more debatable question. The author, however considers the issue quite clear; for the East African Bantu peoples were certainly not in possession of fat-tailed sheep nor of zebu cattle at the time the Hottentots acquired theirs. Their cattle were of Sanga type, derived from an intermixture of Hamitic longhorn cattle and longhorned zebus, whilst their sheep were of the original long- and thin-tailed breed of the Hamites. The sheep of the Hottentots, on the other hand, were fat-tailed animals, and their cattle zebus proper, as the author has shown in previous publications;—not, as Schapera asserts, " of the large, straight-backed, longhorned type (*Bos aegyptiacus*), still found among the native peoples in the Horn of Africa ". The present cattle population of the Horn of Africa, by the way, is also of zebu stock.

Evidence that the Hottentot cattle were of pure zebu stock, and not like the divers Bantu breeds of Sanga type derived from a mixture of longhorned zebus and the original longhorned breed of the Hamites, is furnished by various factors. Characteristic of the skull formation of zebu cattle are the great length and narrow width of the head, the comparatively long, narrow and fine muzzle, the slight prominence of the eyebrows, and the convex profile. These zebu features representing the " desert type " in cattle are found to a full extent in the Afrikander breed descended from the cattle of the Hottentots (Epstein, 1933). Since Hottentot cattle no longer exist, nor any skeletal material, the Afrikander cattle, being of pure Hottentot blood, have to be drawn on for evidence.

Their molar teeth are placed in an oblique position just as in other zebu breeds, the structure of the enamel is simple, the course of the grooves little complicated, and the enamel strongly developed. The back part of the lower jaw-bone ascends vertically in the Afrikander as well as in other zebu cattle, and many other characteristics, by which these animals differ from cattle of Primigenius type, are common to all.

There is another important feature which proves that the Afrikander, and their ancestors, the Hottentot cattle, are to be regarded as real zebus. Practically all zebu breeds are distinguished from other species by the shape of the spinous processes of their dorsal vertebrae the tips of which are bifid. The same phenomenon is found in the vertebrae of the Afrikander cattle, but hardly even a suggestion of such a fissure in cattle of Primigenius type.

In the Bantu cattle, all of Sanga type until the short-horned zebu made its appearance in East Africa a few centuries ago, this feature shows a large range of variation. In some of them the fissure is fairly deep, although not quite as deep as in the red Afrikander cattle and other pure zebu breeds; in others it is hardly perceptible; and even within one and the same breed the differences are considerable (Epstein, 1937).

That there is a similarity between the Afrikander and the zebus brought by Semitic nomads from Abyssinia to Eygpt and other parts of Africa 3,000 to 4,000 years ago, as regards the form and the direction of their horns and all important characteristics of the body, is shown by an ancient Egyptian picture of zebus, dating back to the period of the " New Kingdom ". (See fig. 6.)

This similarity makes it probable that the original characteristics of the zebu are preserved in the Hottentot cattle and their descendents in an even purer form than in zebus of Asia which, as Stegmann von Pritzwald (1924) asserts, were exposed to the influence of short-horned breeds (*brachyceros*).

It is not definitely known when the Hottentots first came into possession of their cattle. It is probable that they received them at a later period than the Nilotic and Bantu tribes did, whose cattle have the old Primigenius breed of the Hamites as a foundation, and at a time when zebus were imported into Africa in such large numbers as to preserve the breed in pure form. Importations to such an extent probably only took place in post-Christian eras, most certainly not prior to the last centuries before Christ.

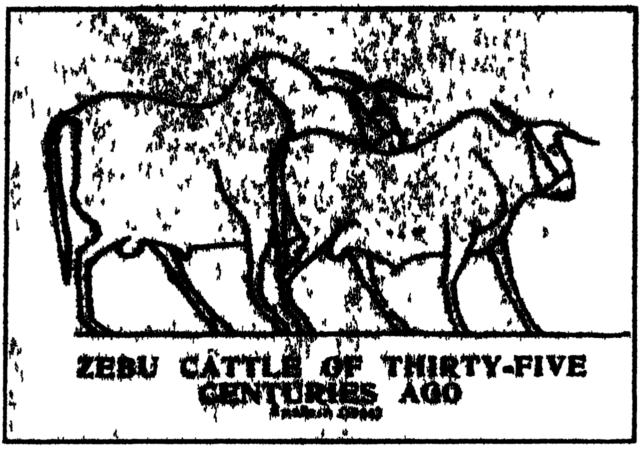
ZEBU CATTLE OF THIRTY-FIVE CENTURIES AGO

Fig. 6.—Zebu cattle of thirty-five centuries ago

According to their own traditions, the Hottentots appear to have come from the lake district of Central Africa whence they were driven out at the end of the fourteenth century or the beginning of the fifteenth by a more powerful race armed with bows and battle axes. But, as Theal (1907) rightly remarks, that was certainly not their earliest home, and it is not even likely they resided there long.

Their zebu cattle must have been obtained in Abyssinia, the starting point of the zebu penetration into Africa, or somewhere in that vicinity; and the people from whom they received them must have been of Semitic or Semiticized stock, since all Hamitic and partly Hamitic races of Africa were originally in possession of longhorned Primigenius cattle or at least of cattle with a preponderance of Hamitic longhorn blood. The only peoples possessing purebred zebus were Semitic immigrants from southern Arabia. Schapera's statement "that cattle and sheep as domestic animals were introduced into Africa by the Hamites" is therefore erroneous in its generalisation. For the role played by the Semites and Hittites in introducing cattle and sheep into Africa is no less important, albeit chronologically later. The distribution of the zebus is as closely connected with the movements of Semitic peoples, as the occurrence of the Egyptian longhorn cattle with the migration routes of the ancient Hamites. The zebus of Africa in general and those of the Hottentots in particular must be considered as but a small branch of the vast family of zebu cattle which extends from the steppes of Central Asia, their original home and place of their evolution, over the whole southern part of that continent from the Pacific to the Red Sea and thence through portions of eastern, southern and south-western Africa to the shores of the Atlantic Ocean.

4. DESCENT AND ORIGIN OF THE HOTTENTOT SHEEP.

Since the Hottentots themselves are relatively new-comers to South West and South Africa, it is evident that their sheep are equally as non-indigenous to southern Africa as their cattle. Nor could their sheep have been acquired in South Africa from the Bantu, as the latter did not then possess fat-tailed animals, but sheep of the long-and thin-tailed Hamitic type.

Therefore we must again follow the migration route of the Hottentots back to the lake district of Central Africa where to this day the sheep population is of fat-tailed type, and further north to Abyssinia and the Horn of Africa whither the Semites brought their fat-tailed sheep from the southernmost point of Arabia.

At the time, however, when the Hottentots acquired their sheep somewhere in the vicinity of Abyssinia or Somaliland, the fat-tailed breed was not as yet so completely established in that region as the zebu. For the sheep of the Hottentots are not of pure fat-tailed stock, but have besides a strain of the long- and thin-tailed hairy breed of the Hamites which followed the migration of their masters throughout Africa.

The Hottentot sheep display features not found in any pure fat-tailed breed. The great variation in the character of their coat, colour and conformation led Hamilton Smith (1827), at a time when far less was known of the Hottentot sheep than now, to divide them into three classes according to differences in the shape of their tail, horns and profile, and according to the colour and condition of their coat. These variations, however, merely show the complete lack of uniformity among Hottentot sheep due to their mixed ancestry. It is impossible to classify them as different types since all variations are connected by a multitude of transitional phases.

The mixed origin of the Hottentot sheep first becomes apparent through the great variety of their coat condition. The majority of fat-tailed sheep of Asia and Africa carry a woolly fleece. But the sheep of the Hottentots are with few exceptions hairy, their coat at the most being a mixture of kemp and wool.

A proof even more conclusive of the diphyletic origin of the Afrikander sheep is the fact that the majority of their rams develop a throat mane. No other breed of fat-tailed sheep is so distinguished. We must give credit for this feature also to the Hamitic sheep among which the rams carry manes.

Further it is necessary to consider the question of the tail formation of the Hottentot sheep. The modifications of tail structure are so numerous and widely divergent that this feature alone is sufficient proof of the mixed origin of the Hottentot breed. Especially the type with a relatively thin, cylindrical tail so tremendous in length as to sweep the ground (sweep stert), clearly points to the influence of the long- and thin-tailed sheep of the Hamites. (See fig 7.)

Further evidence of the diphyletic origin of Hottentot sheep is provided by the shape of their rams' horns. Besides crescent-shaped horns similar to those of other fat-tailed breeds, a large number of Hottentot sheep carries spiral-shaped ones. In some instances the screw of the horn is narrow; in others it is drawn out, its axis pointing outwards. Horns so shaped are not known to have occurred in any pure fat-tailed breed of Asia or Africa, neither in ancient nor modern times. The only screw-horned sheep, except for one or two almost extinct breeds in Sumatra and north-western China, are *Ovis palaeoaegypticus* and some of its descendants. The strepsiceros specimens of the Hottentot breed therefore offer irrefutable proof of their mixed parentage.

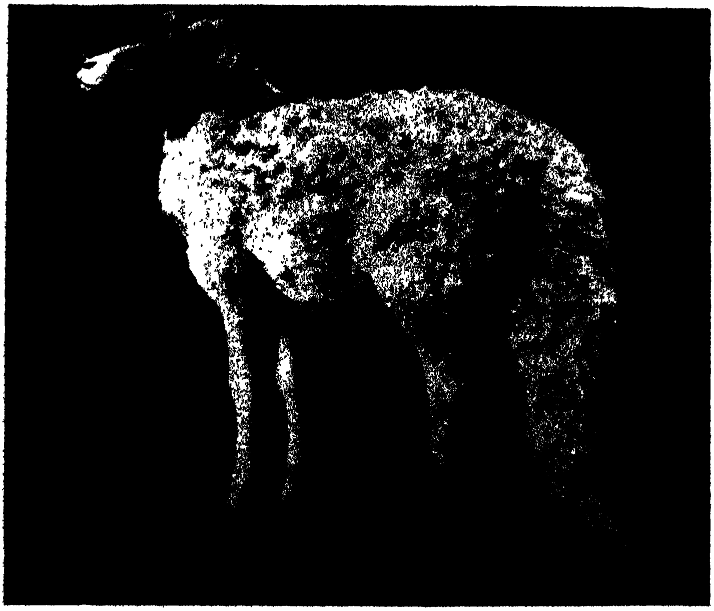

Fig. 7.—" Sweep Stert ". British Museum.

Again, the strepsiceros feature of the Hottentot sheep is reliable evidence that the crossing of the fat-tailed and Hamitic breeds had already been accomplished prior to the Hottentots coming into contact with the Bantu. For the original thin-tailed sheep of the Bantu were not strepsiceros in type, but had crescent-shaped horns.

Before concluding our account on the origin of the Hottentot sheep, a chronological remark may be of interest. We suggested before that the Hottentots acquired their cattle at a later period than the Bantu tribes theirs. Our study of the origin of the Hottentot sheep proves the accuracy of this statement. The Bantu did actually acquire their domestic animals before the Hottentots came into possession of theirs; for just as the Bantu cattle have the ancient Hamitic long-horned breed as a foundation, whilst the Hottentot cattle were of the subsequently imported zebu type, so the Bantu sheep are of the original long-and thin-tailed breed of the Hamites, while those of the Hottentots in addition carry blood of the fat-tailed breed introduced into Africa at a much later date by Semites,

The period of the evolution of the Hottentot sheep in East Africa also corresponds with the above suggestion that the Hottentots did not obtain their domestic animals prior to the last centuries before Christ. For, considering that the fat-tailed sheep of Syria did not enter Egypt before the end of the second pre-Christian millenium, it is evident; in view of the slow means of communication in Arabia and Africa in those ancient times, that it must have taken at least a few centuries more before they arrived from Palestine, by way of Arabia, in Abyssinia and the lake country.

It is even doubtful, discarding the short sojourn of the Hottentots in the lake district of Central East Africa, whether the fat-tailed sheep were established there earlier than a few centuries ago. For it is but fifty years since the first fat-tailed specimens reached what is now Southern Rhodesia from the north (Epstein, in preparation).

That the zebus and fat-tailed sheep were preserved among the Hottentots through so many centuries in their original pure form is due to the fact that the Hottentots were one of the most southern African races of partly Caucasian stock. Therefore they were able to draw away from any pressure from the north and move southwards, where no pastoral tribes, but only Bushman hunters, were dwelling. During their migration to the Cape, their herds of zebu cattle and flocks of fat-tailed sheep were removed from any outside influences, until, at the most southern point of the continent, they met the vanguard of the Europeans.

As we have shown that the Hottentots' domestic animals are rather of Semitic than Hamitic origin, Schapera's suggestion (1930) that "the undoubted Hamitic affinities of the Hottentot languages would incline one to look for a direct Hamitic influence in respect of the domestic animals also", may have to be reversed; that is to say, the undoubted Semitic influence in respect of the Hottentots' domestic animals may incline anthropologists to look for Semitic affinities in the Hottentots.

5. On the Origin of the Hottentot Dog.

An attempt to trace the history of the Hottentot dog has been made by Major Gwatkin (1933-4). But it must be admitted that the difficulties of studying the origin of this interesting canine breed are far greater than in the case of the Hottentots' cattle and sheep. For the Hottentot dog, through lack of interest, or perhaps open hostility on the part of the early settlers, is now entirely extinct, whilst no skeletal material of it exists in any museum. And whereas with the likewise extinct Hottentot cattle we have at least their pure-bred descendants, the red Afrikander breed, for study and comparison, the Rhodesian Ridgeback, though descended from the Hottentot dog and preserving some of its characteristic features, cannot be taken to fill this place. For, as Gwatkin states, " the Rhodesian Ridgeback has undoubtedly been crossed with other breeds introduced by Europeans and certainly in a most haphazard manner ".

The means of arriving at a sufficiently well founded theory on the origin and descent of the Hottentot dog are therefore limited to the conclusions which can be drawn from the few existing pictures of these dogs and from the above-quoted descriptions by Theal and others. Since the Hottentots themselves appear to have originated somewhere between the lake district of Central East Africa and the highlands of Abyssinia, in the same region where they acquired their cattle and sheep, it is reasonable to assume that they came into possession of their dogs in the same place. The descriptions and pictures of the Hottentot dog show that it belonged to the pariah type found practically throughout the whole of Africa and the East.

The Hottentot dog, however, has one peculiarity which distinguishes it from other pariah breeds:— the hair on its spine points forward. And this feature has given rise to a strange theory on the Hottentot dog's origin. Gwatkin (1933-4),namely, has discovered that the Hottentot dog is not the only breed whose hair curls forward on its spine, but that it shares this peculiarity with a dog from the small island of Phu-Quoc in the Indo-China seas. This dog is described as follows:— " A long head with powerful jaws, erect ears, reddish eyes, with a savage expression, somewhat coarse body, neck very long and flexible, shoulders sloping, belly drawn up, loins broad and strong. Straight and lean legs, stifles rather straight with muscular thighs, longish feet with hard pads. Coat, on the whole body and legs very short and dense, on the back the hair is growing the wrong way, towards the head, and is much longer and harder. Colour, reddish fawn with black muzzle, the hair on the back being darker. Height, about twenty-one inches, weight about forty pounds".

In the absence of an osteological analysis of thePhu-Quoc dog's skull, the description points to atypical southern Asiatic pariah dog, a form closely approaching the dingo, but with a distinction in the character of its coat, namely, the hair along the spinal column growing forward. From the similarity of this feature in the Hottentot and the Phu-Quoc dog Gwatkin (1933-4) argues that " the Hottentot dog, if not actually the same as the Phu-Quoc, is definitely derived from the same Asiatic stock that originated the Phu-Quoc".

But whilst it may hold true that the African and southern Asiatic pariahs are descended from the same wild canine, Gwatkin's argumentation is nevertheless erroneous. For even if one of these two breeds had the hair on its back lying normal, this would not contradict the theory of their monophyletic origin. On the other hand, the similarity of this feature in the Hottentot and the Phu-Quoc breed provides no support whatsoever for this theory. The Incas, at the time of their country's conquest, possessed bulldog-, dachshund- and terrier-like breeds. Yet it is well-known that the

Inca dogs were derived from entirely different wild ancestors than are the Old World bulldogs, dachshunds and terriers. This example merely shows that the range of mutations due to the effects of domestication is limited; even entirely different species sometimes showing parallel mutations, such as hairlessness occurring in humans, dogs, swine, cattle, mice, goats; strepsiceros horns in the ancient Egyptian sheep and goats; woolliness in sheep, poodles, some Russian and Paraguayan horses, certain south-eastern European pigs, Angora cats, rabbits and Guinea pigs; tags or lappets in sheep, goats and pigs; ankylosis of the caudal vertebrae in sheep, dogs, cats and mice; coat colour, long pendent ears, fat deposits, hornlessness, and many more of the like. And such parallel mutations or convergencies may be induced in domesticity by the same or by different causes. In the case of the Hottentot and the Phu-Quoc dog the cause of the convergency in coat character may have perhaps been the same. We could imagine, for instance, close inbreeding, as both these dogs are insular forms, so to speak, the Hottentot dog being long removed from any foreign influence, since its masters constituted one of the most southern African races of partly Caucasian stock, in a country devoid of people possessed of domestic animals. Under such insular conditions it is relatively easy to preserve and propagate spontaneous mutations and atavistic Mendelian factors.

The parallelism in the character of the coat in the Hottentot and Phu-Quoc dogs therefore proves absolutely nothing with regard to their ancestry; and it is a fallacy to believe that this mutation could not have been developed in Africa spontaneously, but that the ridgeback, " favourite of the navigating Easterners ", must have been introduced by sea from the original home of the Phu-Quoc. Gwatkin goes so far even as to state that as the Chinese and other Eastern nations were navigating the Indian Ocean for a considerable period before the 10th century, it was from this source doubtless that the Hottentots received their dogs. In support of this theory he even mentions Mongolian characteristics in the Hottentots themselves.

6. Land Tenure.

Among the Hottentots every tribe had its own territory which was the tribe's communal property. Passers-by or new-comers had to get the chief's permission if they wished to hunt on a tribal holding or pasture their cattle and sheep there. In the early days of the Dutch settlement the different Hottentot tribes were situated far apart, each tribe holding certain centres round which it migrated, and claiming as its territory all land where its members were accustomed to graze their stock (Schapera, 1930). Since the Hottentots depended almost entirely upon milk for subsistence, they needed a large number of cows and ewes, and consequently a great extent of pasture for each separate community (Theal, 1907). This was the cause of their being so thinly scattered. For, owing to the natural poverty of their country and the frequent periods of drought, they had to move about freely in search of pasture. In the early days the boundaries of the tribal territories were not very clearly defined, and on that account fights for pasture land occured quite often. However, as long as sufficient grass and water were available for all, there was little objection raised as a rule to intrusion.

Along the west coast of southern Africa the land of the different tribes appears to have been even less clearly demarcated than in the vicinity of the Cape. But owing to the aridity of this region particular value was attached to the possession of the wells and waterholes which the Hottentots dug in the beds of the periodical streams and covered over carefully to prevent evaporation (Stow, 1905). This does not mean, as Mrs Hoernlé (1918) points out, that strangers could not use the water, but that certain specific tribes had a prior right to different fountains or pools, established by habit or through their work in digging such wells.

In later times, when the pressure of the Dutch from the south and of the OvaHerero from the north restricted the free movements of the Hottentot tribes, tribal boundaries were more carefully defined between the chiefs, and any encroachment was forbidden and deeply resented. If a tribe wanted to move into the territory of another, permission had first to be asked. If they were on friendly terms this was often granted without levy, but if relations were strained owing to previous cattle raids, permission was refused or a tribute of heifers demanded as payment or acknowledgement of the resident tribe's ownership of the district. Of course, sometimes permission was refused, and if encroachment took place nevertheless, it led to war, It often happened that a tribe paid the tribute for a short period, but refused further payment when once settled down and accustomed to its new surroundings, even under threat of war.

Communal land of a tribe could not under any circumstances become the property of an individual or the chief's. It was regarded as inalienable. The chief had no right to sell such land, and even the granting of usufruct was subject to the consent of every family under his rule, among whom tributes or purchase moneys had to be equally divided, (Wandres, 1909).

Every Hottentot had a personal right to the use of his tribe's land, water and grazing for himself, his family and his stock. No chief could interfere with or could deprive a subject of such rights. A first-comer had prior rights. If a man dug a well or opened up a spring, it was his property and he retained the sole right to it. Anybody wishing to use such water for himself or his stock had first to ask permission from the owner.*

Every Hottentot was allowed to move freely over the tribal territory and to erect his hut wherever he pleased, without restrictions from the chief or anybody else. But it must be understood that his right was merely to the use of the land and in no case implied full ownership to the exclusion of others.

In certain respects, however, members were limited in the unrestricted exploitation of the tribal land. For example, the chief could order certain grazing grounds to be vacated so they could be rested. Such orders were in all instances implicitly obeyed by the people. The consent of the chief was also necessary if the inhabitants of a kraal wished to burn the veld in winter so as to speed up the growth of the young grass following the first spring rains. These instances show that despite their nomadic life the Hottentots had a certain knowledge of pasture management gained from experience.

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* Report on the Natives of South West Africa and their Treatment by Germany. London, 1918, pp. 75-76.

7. OWNERSHIP AND ACQUISITION OF CATTLE AND SHEEP.

In the early days of the European settlement at the Cape the Hottentots were extremely rich in domestic animals. Every family had its own cattle and sheep; and although it was customary for those owners who dwelt as a community to graze their cattle and sheep together, the latter were not common but private property. To quote Kolben (1719): " There is only one herd of horned cattle in each kraal, but this does not belong to one person alone, such as the chief or one of the wealthier members, but every inhabitant of the kraal has a share in it, be it large or small." The cattle were considered so much the property of the husband and wife that the former could not dispose of any without the consent of the latter. Some of the cattle were killed entirely for the women's use. It was rarely that men and women ate of the same ox or cow (Stow, 1905).

Every Hottentot knew the animals he possessed. To facilitate discriminating between the stock of one owner from his neighbours', it was common practice among them to select their breeding stock according to colour. As a rule, every stock owner kept and bred animals only of one and the same colouring, exchanging those that differed for animals of the desired colour (Schultze, 1907). Schultze also mentions that the Hottentots marked their cattle in special ways by cutting, perforating or lopping their ears.

Cattle and sheep were acquired in different ways:— After the death of a Hottentot his stock was divided among his heirs. But in a wealthy family the father provided his children with stock whilst still alive. Usually a newly born child had some animals set aside for it, and at the puberty ceremonies or marriage both sons and daughters were given a few head of stock by their parents. Cattle were further acquired by barter with such objects as milk pots and weapons, and in more recent times European trade goods (Schapera, 1930).

Those Hottentots who were not given stock by their parents used to serve the wealthier owners as herdsmen, receiving a cow or a ewe for their service; or else they left the kraal for three, six or twelve months to work on a European's farm. In this manner they managed to acquire in due course a few head of cattle and sheep. It was easier for them to obtain stock from Europeans in this way than from their own people; for on a European's farm they used to get their weekly ration of tobacco and dagga, and sometimes a little money as well. Besides, they never accepted oxen or hamels for their work from European farmers, but only breeding cows or ewes. It was far more difficult to obtain cows and ewes from their own tribesmen, since the latter were extremely reluctant to part with breeding stock (Kolben, 1719).

Another common method practiced among the Hottentots was the herding of another's stock in return for part of the progeny, A wealthy Hottentot would place some or all of his stock under the care of an impoverished neighbour, allowing him to use the milk for his own nourishment and to take half the herd's increase. Even young boys would hire out their services as herdsmen to wealthy stock-owners, receiving at first an annual payment of one or more sheep, according to the size of the flock and herd, and later on a third or a half-share of the progeny. With good management and luck a good herdsman could in a number of years acquire quite a fair-sized herd of his own in this way, especially since the Hottentots very rarely killed cattle or sheep for food, and never female breeding stock, if they could help it.

Some shepherds remained with their masters for life, even though they had accumulated fairly large herds and flocks of their own. These their own children looked after, whilst they themselves herded their masters' stock (Schapera, 1930). The majority, however, having earned a few head of stock in the service of Europeans or wealthy tribesmen used to return to their own kraals to look after their cattle and sheep for the rest of their lives, bent on increasing them by every means possible. For a rich man was always listened to and had more influence than the chief himself (Mossop, 1935).

Finally, the foundation of many Hottentots' prosperity was laid by cattle-raiding among other tribes which was a common practice in the past. Their battles almost always took place in the vicinity of their cattle kraals. We have, however, no definite record as to how cattle and sheep so obtained were divided among the members of a raiding party.

8. HERD AND FLOCK MANAGEMENT.

As in the arid country of the Hottentots water was found at only a few places, these usually served as sites for their encampments. At such places the Hottentots remained for as long as the pasture in the vicinity was sufficient to sustain their herds. During the rainy season, when certain pools were temporarily filled with water, they were able to move about more freely, availing themselves of pastures that were useless to them during the dry season.

In the old days the encampment was in the form of a great circle enclosed by a thorn fence. The huts were erected round the circumference facing the great open space in the centre which served as a fold for the stock by night. Special enclosures were made for the calves and for the lambs, but there were no enclosures for mature cattle or sheep, these just lying in front of their owner's hut till driven out to pasture at morning (Schapera, 1930).

The animals were pastured in the vicinity of the kraal. The various owners did not appoint a special herdsman to do this work, nor did they go all out to look after the herds and flocks, since this, as Kolben says, would not at all suit their lazy lives. Every day, according to the number of stock, one, two or three herdsmen were sent out from amongst the kraal inhabitants to look after the animals. The herdsmen changed daily, and nobody was freed from this service. If a wealthy Hottentot did not wish to do his turn, he had to send a servant or relative in his stead.

The oxen did not need any special care in herding since they were only required occasionally. They used to rove at large, returning about every two or three days to drink at the watering places; during the cold season staying away even four or five days at a time. When wanted for work they could easily be secured at the water; otherwise, they rested in the vicinty of the pools during the heat of the day, proceeding to the veld when the cool of the night set in. If an ox had to be fetched from the veld, the Hottentot sent out to look for the animal, easily recognised the footprints of his own herd amongst the numerous tracks crossing the veld in all directions, and from between the tracks of his herd those of the particular beast he was after.

Cows too old to calve and kept for slaughter were sent out with the oxen. Since they went further away from the kraal than the milch cows, to pastures less overstocked, and did not lose so much time in being driven to and fro, they soon became fat. Young sterile cows being usually in good condition and having a glossy coat were left in the herds, simply to please the eye (Schultze, 1907).

Of course, the cattle remained in the open over night only where not in danger of attack by beast of prey. In the early days, when lions and other wild animals still abounded, all cattle were kraaled at night as lions and leopards used to play havoc with the Hottentots' herds and flocks, especially on dark nights.

No attempt was made to control the breeding of cattle. The bull was allowed to stay with the herd. At the end of the rainy season, i.e. March or April, when grazing was plentiful and the cows in good condition, the bull followed close on their heels serving them when they came into season. At the beginning of the dry period the bull left the herd and roved alone in the veld until the next breeding season, only coming to the common water pool when there was not enough water elsewhere. His movements were quite unrestricted, and everybody kept out of his way since his wild, lonely life made him savage and dangerous to both man and beast.

Most of the Hottentots did not milk their cows in the morning because, as Mr. Sass, a missionary who resided for some time amongst them, states, their rest would be disturbed by early rising (Stow, 1905). After the calves had been allowed to suck, the cows were driven to pasture and left to graze until the herdsmen considered it time to drive them home or the attraction of the young calves brought them to the kraal of their own accord at the fall of evening. It was the herdsmen's prime duty to protect the cattle against attacks of lions and other wild beasts. The Hottentots struck the attackers with javelins or heavily poisoned arrows surely aimed (Dapper, Ten Rhyne and Grevenbroek, 1933). Usually boys were charged to watch the herds and flocks during the day, unless an attack from enemies was feared, young men then assisting (Stow, 1905).

The calves were driven to pasture in a different direction from the cows. Here they remained under the guard of the herdboys who drove them home at evening. Sick calves and those growing too thin from lack of milk were allowed to remain with their dams day and night.

When the cattle had returned from the fields, they were watered and driven into the camp enclosure where each beast was tethered to a stake by its left forefoot. The calves were then allowed to run to their dams, and at great exertion the Hottentots bestirred themselves to rise and milk them. After the milking the calves, having sucked the udders dry, were locked up for the night together with the young lambs in a hut specially built for the purpose or, as Schultze states, in an open kraal the walls of which were made of thorn bushes or, more rarely, of stones and clay. Older calves were tethered to stakes in front of the hut to prevent them getting to their dams or from falling a prey to wild beasts. The cows were tethered in a circle and fastened to one another by ropes made from reeds. The sheep were kraaled in the centre (Kolben, 1719).

Occasionally, if the pasture round the encampment was overstocked and poor, the cattle and sheep were driven far into the veld and kept at outlying posts for weeks and even months under the care of a few herdsmen (Schultze, 1907). Wealthy Hottentots sometimes had several cattle posts which were inspected periodically by the owner himself or his chief herdsman. As a rule, however, each family remained and moved with its own herd and flock, sometimes even acting independently of the rest in search for grass and water (Schapera, 1930).

When shifting the encampment to another place, the young boys had to get up early in the morning and drive the calves and sheep slowly along so that they had time to graze on the move, and that the young lambs could follow. The main herd followed later at a quicker pace; and as the cattle were not allowed enough time to graze on the way, they were left in the open during the night following.

The sheep, like the calves, were herded by day in the veld and kept at night in the kraal. They were under the care of herdboys who guarded them from danger and saw to it that they did not spread too widely over the veld. They had also to be collected during the midday heat under some trees so they could rest in the shade. Young lambs remained all the time in a special kraal and were only allowed to their mothers at morning and evening milkings. The lambs were weaned by rubbing the ewes' udders with dung. Once weaned they were pastured together with the ewes.

The rams always went with the flock; and whilst the Dutch settlers, as Kolben mentions, used to separate the rams from the ewes after the mating season, because they did not want the ewes to lamb twice a year, thinking that both ewes and lambs would grow too weak, the Hottentots did not follow this practice. They left the rams together with the ewes the whole year round thus obtaining two lamb crops annually, one or two lambs a time. Yet, Kolben (1719) writes that he could find no difference between the sheep of the Dutch and those of the Hottentots.

Calving time was usually in December and January; but the irregularity of the rainfall and the consequent scarcity of grass often retarded the mating and calving seasons by weeks or even months.

If a cow showed signs of being near parturition a special herd-boy had to watch over her and bring dam and calf home as soon as they could walk. The colostrum was considered unwholesome for the calf, because the Hottentots believed it turned into a hard substance in the calf's stomach. They themselves liked it all the more drinking it boiled or mixed with ordinary milk.

The milk production of the Hottentot cows was adapted to the needs of their calves. As soon as the latter found enough sustenance in the veld, the milk flow of their dams ceased. During periods of drought the cows gave so little milk that their calves often died of hunger, even if the dams were not milked at all.

The Hottentots followed the growth of their herds and flocks with the greatest interest remembering the life history of each animal. They knew their animals' ages better than their own and were able to tell whether a heifer was pregnant for the first time or had already had a calf, and how many times each individual cow had calved. For all these stages the Hottentots employed special terms; so also for cows in milk or in a dry state.

Formerly they designated their domestic animals according to their colour or physical development. Schultze (1907) records nineteen such terms in use among the Naman for cattle, six for goats and three for sheep.

None of the Hottentots ever exchanged a white ox or cow which they looked upon as an invaluable leader of the herd (Dapper, Ten Rhyne and Grevenbroek, 1933). This preference for white cattle is found among many Hamitic and Hamiticized peoples. The ancient Egyptians, for instance, worshipped Isis in the likeness of a white cow, and used to pray that the Lord (Great Chief) should send them a white ox. The half-Hamites of East and East Central Africa slaughter a white bullock at their great ceremony of handing over country from one age-grade to the other (Seligman, 1930). The Zulus have amongst their cattle a type with white bodies, black muzzles and black insides to the ears, which at one time were regarded as royal cattle, and were treated with great respect, if not reverence; in fact, it is thought that to some extent they were held as sacred.* But this reverence for white animals is not only found amongst peoples of Hamitic stock nor limited to cattle. Many other primitive peoples held white domestic animals as sacred. Among the ancient Germans and the great majority of the Slavs, for example white horses were consecrated to the god of war, whilst in Siam white elephants (albinos) were held as sacred (Rheinhardt, 1912).

Surplus bulls and rams were castrated by the Hottentots. There was one man in each kraal who used to do this work for the whole encampment. Young bulls were gelded when about a year old.

' Information supplied by Mr. E. Wyatt Sampson.

The testicles were not excised since this operation was regarded as too dangerous. The calf was thrown onto its back, its feet fastened with ropes, and its head held to the ground. Then the scrotum was bound closely and tightly with a thin, elastic thong made of antelope hide, so as to cut off communication with the spermatic vessels, the calf then being allowed to run in that condition till the testicles dried up and fell off (Kolben, 1719).

Oxen castrated at maturity were called by a special name designating strength because such animals are indeed stronger than oxen gelded during calfhood, although far less hardy (Schultze, 1907).

Rams were castrated when about six months old, or when too old to be of service. They were thrown onto a flat stone, and the scrotum was tied up in the same manner as bulls'. The operator then took a round stone and beat the testicles to pieces. The wound was left to heal by itself (Kolben, 1719).

The successful castration was celebrated by a feast consisting of the meat of a specially-slaughtered calf cooked together with the pounded testicles.

Ordinarily, the Hottentots did not slaughter any cattle or sheep for food, except those which, owing to sickness, old age or lameness, were unable to follow the herd. This rule was only broken when preparing their ceremonial meals. On killing a sheep, the Hottentots always looked for the leanest in the whole flock because, as Sparrman (1789) writes, the rest were intolerably fat. But since the Hottentots in reality were extremely fond of sheep's fat, it is far more likely that their preference for the leanest sheep in the flock was due to the fact that they knew from experience that worms or disease were the usual causes of exceptional leanness among their fat-tailed sheep, rendering the animals from a breeding point of view or for other reasons less valuable.

In the protection of their herds and flocks from wild animals, especially jackals, the Hottentots were assisted by their dogs which were extremely watchful and gave immediate warning of the approach of strangers or beasts of prey by loud barking. By means of milk the Hottentots familiarised the shy animals with the herds; and with their own persons by carrying a piece of rawmeat for a few days in their veldshoes, afterwards giving it to the dog, or by applying sweat from their armpits to the dog's nostrils.

Bitches which they wished to prevent from getting in pup were branded with hot irons around the vulva. This made them so sensitive at these parts that they refused mating.

In general, however, dogs were never cared for properly and much less valued by the Hotentots than among Bantu peoples who are extremely fond of dogs. They used to carry this passion to such a height that if a dog particularly pleased them, they would give two bullocks in exchange for it (Paterson, 1789).

9. MILKING AND THE USE OF MILK.

The milking of cows and ewes was performed by women, in the same right-sided manner as practised in Europe. The stores of milk were under the control of the women, not under that of their husbands, as with the Bantu among whom milking is essentially the task of the men, the women having very little indeed to do with the cattle, being ritually prohibited from all contact with them (Schapera, 1930). The Hottentot men tended the cattle, but their women did the milking (Theal, 1907).

It is not by chance that among the Hottentots the women, and not the men, milked the cattle and were complete sovereigns over the cows and milk. For among Semitic peoples likewise it is the women's task to milk; but throughout the entire group of Hamitic and Hamiticized tribes, with but one or two exceptions (Ovaherero), this work falls to the men. The cultural importance of this custom is illustrated by the fact that the Eastern Hamitic Beja actually despise the Arab tribes in their neighbourhood for allowing their women to do the milking (Seligman, 1930).

Unless their calves were present, the cows of the Hottentots, like all native cattle south of the Sahara, did not allow themselves to be milked, but kept their milk back Even when the calves had sucked the first milk and were present at the milking, the cows did not yield all their milk, not permitting themselves to be stripped, but retaining the last rich milk for their calves.

Therefore the Hottentot women first allowed the calves to suck a little, then, having bound the cows' hind legs together, proceeded to milk them; but the last milk they again left for the calves. If a calf had died or been killed they placed its skin on another calf, and then began milking. But when the skin was not in their possession, they tried another method of inducing the cow to part with her milk: They bound her hind legs with a thong, so that she could not kick, and blew air into her vulva. In most cases this had the desired effect. The assistance of men at this performance was permitted (Kolben, 1719). Keller (1894) observed the same method among the Somali, but with this difference that the latter blew air into the rectum of the cow. (See fig. 8.)

The teats of the cows and the hands of the milkers were lubricated with milk. Only during the first days after calving the colostrum was not allowed to come in touch with the teats. During these days the teats and the milkers hands were moistened with saliva (Schultze, 1907).

Baskets of a peculiar kind were used for milking, composed of roots plaited together so closely that they could hold not only milk but even water. These baskets were never cleaned, the milk being encrusted upon them. The milk was not strained, but kept in the same basket into which it was milked, contaminated by hair, dust and manure. (See fig. 9.)

Fig. 8.—Hottentots inducing a cow to part with her milk. After Kolben.

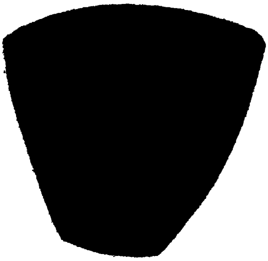

Fig. 9.—Hottentot milk basket. After Sparrman.

The use of basketry vessels for milking seems to have been a sacral custom among the Hottentots at one time, if it is possible to take the practices of other African native peoples as evidence. Even to the present day none of the Eastern Hamitic Beja tribes, for instance, milk into a clay vessel or will put milk into these, in spite of the fact that many of them make good pots, nor would it be permissible to milk into the tin vessels introduced by European trade. Basketry vessels so well made as to hold milk without any tendency to leak are considered the appropriate receptacles (Seligman, 1930).

According to Kolben, the milk was either drunk fresh or sour, or after having been cooked together with edible roots. Sometimes it was mixed with a vegetable substance, such as the green leaves of the ebony wood, which were chewed and spat into it, or the sap of the acacia (Schapera. 1930). Any surplus was exchanged for tobacco or churned into butter. Sparrman, on the other hand, was informed by the Hottentots that sweet milk was unwholesome. and that they never ate it until curdled. They always mixed it with sour milk in a leather bag which could contain about six gallons and was made from an undressed skin taken off entire, with the hairy side turned inwards. The sour milk mixed every day with some fresh was often preserved for many weeks without turning bad (Sparrman, 1789). The bag in which it was kept was never cleaned and commonly in a filthy state as indeed was everything else in and about the huts (Theal, 1907). Other types of vessels used for preserving milk were wooden pails and earthenware pots.

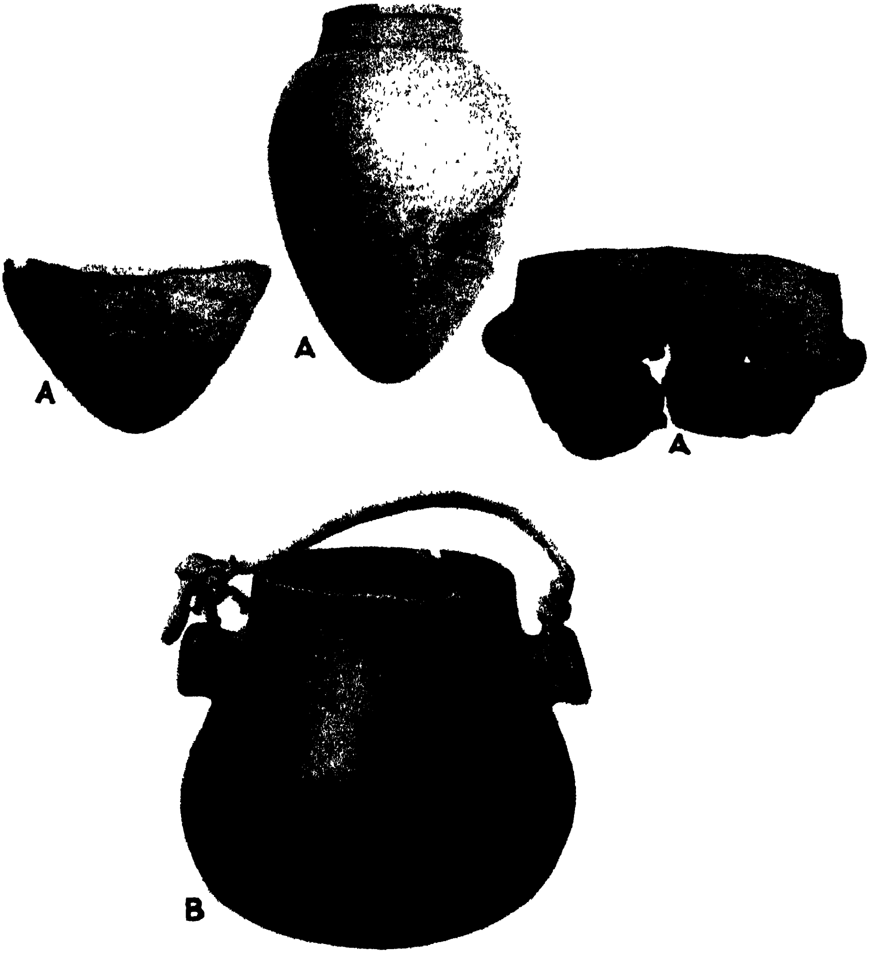

Fig. 10.—Clay Milk Vessels of the Hottentots.

(*a*) Albany Museum, Grahamstown. After Schönland.

(*b*) After Schultze.

The leather bag was made in the following manner (Schapera, 1930): A freshly killed goat, calf, gemsbok, or steenbok was cut open all along one side, from the left foreleg right up to the shoulder. Through this opening the whole body was taken out from the skin, the head and legs having first been cut away. Then all the openings were sewn up, with the exception of that of the right foreleg, which served as a spout for pouring the liquid in and out, and had a stone cork which could be tied fast. The fresh skin was turned inside out, the innerside cleaned of all particles of flesh and fat, and then dried, the hair remaining on the inner side of the bag.

The most common form of earthenware milk vessel was a large-bellied urn with a narrow-rounded base, small mouth and two ears through which a cord could be passed in order to suspend the vessel. Others were quite cylindrical, with almost flat bases, whilst in a third type the base was slightly rounded and larger than the rest of the pot. All Hottentot pottery was unglazed, though as a rule quite watertight (Schönland, 1903).

The pots were made by the women, each family making its own. The clay was taken from termite heaps, and kneaded together with the ants' eggs. After having been modelled to the required shape, it was smoothed inside and outside by hand, and dried for a couple of days in the sun. Then it was put into a hole in the ground and, with a fire inside and out, was burnt until hard (Kolben, 1719). The potter's wheel was unknown to the Hottentots before the advent of Europeans. (See fig. 10.)

Their wooden pails were made by hollowing a block of wood, cutting and boring it, no fire or other hot objects apparently being employed. The block was shaped and smoothed on the outside with a knife, and hollowed out with a semicircular iron blade fixed in a wooden haft. Some of the wooden milk pails resembled the earthenware pots in shape, even having similar ears for suspension (Schultze, 1907). (See figs. 11 and 12.)

The trees used for this purpose were soft woods and thus easily worked, besides showing less tendency to crack when dried or exposed to the sun. The Bantu usually took their wood from *Sclerocarya caffra*, *Cussonia sp.*, *Burkea africana*, *Schotia transvaalensis* or *Schotia brachypetala* (Curson, Thomas and Neitz), whilst the Hottentots used chiefly Acacia wood. Two wooden kitchen utensils of the Hottentots, reproduced by Schultze, were made from wood of the Ana (*Acacia albida* Dél.) and the Kameeldoorn (*Acacia giraffae* Burch.) respectively.

To make butter the Hottentots used a bag sewn from a dried skin, with hair turned inwards. Into this dirty hairy bag they poured the milk, tying the opening up with a strap of leather. Two men or two women, or a man and woman, then took the bag by its two ends, shaking the milk rapidly until it turned into butter. Occasionally the freshly-cut thick roots of a certain plant (?Portulaca sp.) were put in with the milk to increase the yield of butter (Schapera, 1930).

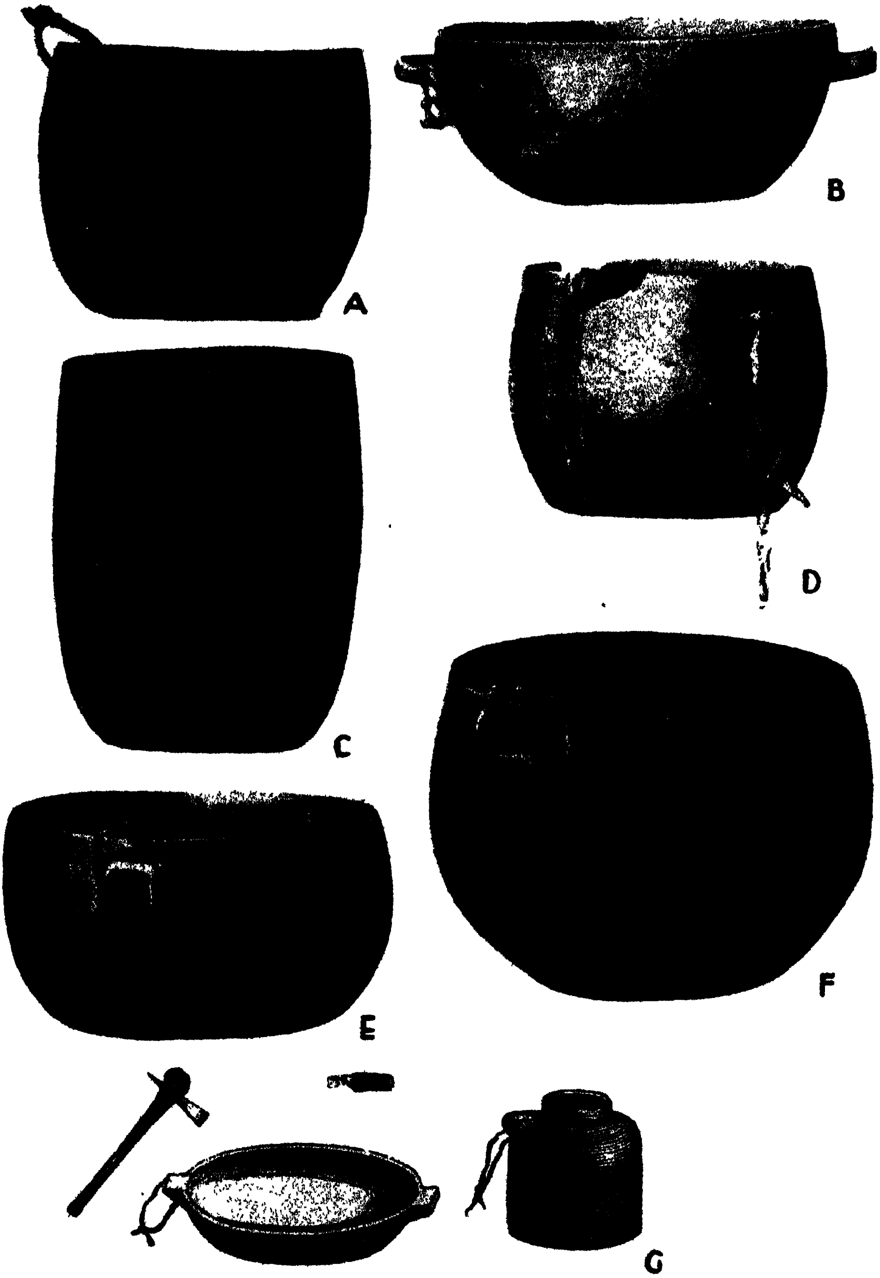

Fig. 11.—Eared wooden milk vessels of the Hottentots.
After W. Burchell and L. Schultze.

The butter, once formed, was taken from the hairy bag and put into an empty pot, unwashed, just as it came out multicoloured by manure, hair and other dirt.

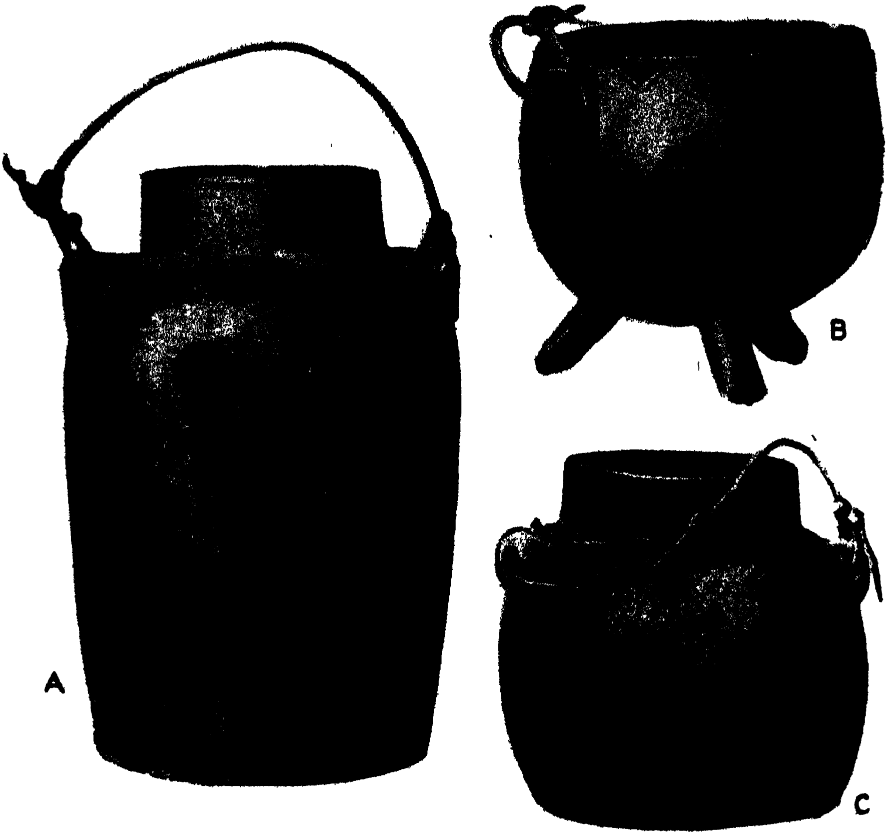

Fig. 12.—Wooden milk buckets of the Hottentots. After L. Schultze.

The butter-milk was either given to young lambs and calves, or calves, or drunk by the Hottentots themselves who used to be quite indifferent to the dirt passing down their throats along with the milk.

Milk of cows was never mixed with that of ewes, but the two were kept separate. For cows' milk could be drunk by men and women alike, by young and old; but men and boys were not allowed to partake of even a drop of ewes' milk, which was left solely for the women. Kolben reports that at his time the Hottentots were no longer aware of the reason for this custom. All they knew was that their parents had told them that ewes' milk was unwholesome for men and its use therefore forbidden them (Kolben, 1719).

Children were taught to suck the ewes, very often deriving their whole sustenance from this source (Theal, 1907).

The special regard of the Hottentots for milk is one of their most interesting characteristics and which on analysis can be shown to go back to an old Hamito-Semitic civilisation.

10. Fighting Oxen and Beasts of Burden.

The great interest the Hottentot pastoralists took in their cattle and the latter's importance to their masters can be gauged from the following particulars quoted from the interesting work of P. Kolben (1719), who visited the Cape in 1705 and stayed there for a period of eight years.

" The Hottentots ", he writes, " have a sort of oxen which they call Backeleyers or fighting oxen. These are the biggest, strongest and boldest animals of the whole herd. In each kraal there are about five or six, and in some even more. They are chosen from the herd by old Hottentots who know how to instruct them. The Backeleyers are of great use, too, in the controlling of the herds at pasture; they have to fetch in stragglers, protect them against beasts of prey and bring the herds within compass."

" In the Hottentots' wars against one another these Backeleyers create havoc among the enemy. They gore and kick, and even trample to death, with incredible fury. Victory is assured to whichever side commands the largest number of fighting oxen."

" A stranger, especially a European, approaching a herd, must be on his guard; for these Backeleyers, which generally feed on the outskirts of the herds, quickly discover him and make for him at a full gallop. And if he is not within earshot of a Hottentot and has no means of escape, he stands in great danger. Sticks or stones thrown make no impression on Backeleyers, which go straight for the intruder. If, however, the person attacked shouts for assistance, and is heard by a Hottentot, the latter will whistle through his fingers, and the ox will stop, look doubtfully at the stranger, follow him towards the Hottentot, and then return leisurely to its herd and pasture."

" If no Hottentot is within hearing to come to his assistance, there is nothing to do but to shoot. Frightened by the report of the gun the Backeleyers run away. But if he has no weapon and no tree near by to climb, he has to wait in fear for what will happen. Even if he is not killed outright, he will be gored and kicked so badly that he will suffer serious injuries, perhaps fatal."

" I have often been chased by Backeleyers myself. When I saw them making for me, and no Hottentot was near to hear me shout and come whistling to my assistance and arrest their attack, I have been obliged to discharge my gun. In such circumstances I have always questioned the Hottentots as to the manner in which they instruct their oxen, and have learned from them that a young ox is fastened to an old Backeleyer and taught by blows and other means to follow him. At night they are also tied together so that the young ox is always close to the old one."

" The oxen used for carrying purposes are likewise very strong and stately beasts chosen from the herds by old men skilled in the judging and training of cattle. At about the age of two years an ox destined for a baggage animal is selected, caught and thrown on its back. Its head and feet having been fastened with strong ropes to stakes firmly fixed in the ground, one of the Hottentots takes a knife and pricks a hole through the beast's upperlip, between its nostrils. Into this hole he puts a stick, about an inch thick and a foot and a half long, with a hook at the top to prevent its falling out."

" By means of this hooked stick it is very easy to break the ox to obedience; for if he refuses to be governed and tries to run away, the Hottentots fix his nose by the hooked stick to the ground, and this torture makes the ox tractable; when a Hottentot takes an ox by this stick, the animal follows patiently in terror of pain. When teaching him to carry burdens on his back, his nose is again fixed to the ground by the stick, or the latter held by a Hottentot, until the beast ceases to be refractory."

" When the Hottentots decide to break up their old kraals and move to new places, their huts and cooking utensils are loaded onto the backs of their oxen. Those not needed for transporting the huts are saddled with double baskets made from sticks and strips of leather, in each of which is room for two old or sick people."

"There are usually far more baggage- than fighting oxen attached to a kraal, but their relative numbers depend on the size of the tribe and its requirements."

This is Kolben's account.

Over a hundred years later, Hamilton Smith, (1827) referring to some Hottentot cattle of extraordinary size, writes:— " It is from these that their Backeley or war oxen are chosen: they ride them on all occasions, being quick, persevering, extremely docile, and governed by the voice of a whistle of the owners with surprising intelligence. They thrive most in the Zuure Velden."

The riding oxen were guided by a bridle of raw hide, attached to a primitive bit of wood or leather passed through the cartilage of the nose. Instead of a saddle a sheepskin was thrown over the back of the animal and fastened by a rope drawn tight round the forepart of its body. No stirrups were used, but both men and women rode the animals with ease, being accustomed to do so from childhood (Schapera, 1930).

Description of the training of pack- and riding-oxen are given by Schultze and Von François:

The Hottentot charged with the catching of a young untrained ox made his way through the herd and placed the loop of a long rope by means of a stick between the forelegs of the animal wanted, so that the ox could be caught by one of its legs when moving along. Four to six men held the other end of the rope waiting until the animal had calmed down a little. Then two men quietly approached the ox from behind, one on its right, the other on the left, drawing another long rope over the back of the ox until it reached the horns. One of the two next made a large circle around the animal's head forming a loop into the rope which was drawn tight round the horns. By means of these two ropes, the one around the horns, the other round one leg, the ox was pulled to one side, whilst another Hottentot twisted its tail over the animal's back so forcibly in the opposite direction that the beast was thrown to the ground giving up all resistance (Schultze, 1907).

Von François' (1896) description differs slightly: An ox was given its first training by young boys when about twelve to eighteen months old. The animal was caught with a loop round one of its hindlegs, a few boys holding the leg by a rope, whilst others put a thong round the animal's horns, pulling the two ropes towards one side. A third rope was fastened round the body of the ox, and one of the boys jumped on the animal's back holding on by this rope. As a rule, the ox immediately started to bellow and tried to break away, sometimes succeeding in throwing its rider off. But the ropes fastened to its horns and leg prevented the animal from getting away. After many futile attempts the ox became tired and refused to move from its place. At this moment the boys started to thrash the beast with leather thongs or to bite into its tail. The ox once more tried to break away, but soon stood still again when realising that all attempts were in vain. Then the game started all over again. This training having been repeated for eight days every morning and evening for about half an hour, a piece of wood was passed through the cartilage of its nose, a string being fastened to its two ends serving as a bridle. For a time the young ox was fastened to the horns of an old experienced riding beast, and the pupil began to walk patiently at its master's side.

"One can travel comfortably and securely in this way for a great distance and over rugged mountains." says Mrs. Hoernlé (Schapera, 1930), "provided one is not in too much of a hurry."

11. PREVENTION AND TREATMENT OF ANIMAL DISEASES.

Since the Hottentots depended almost entirely upon their cattle and sheep for subsistence, especially during the dry summer, the health of their stock was of the utmost importance to them, as was fully reflected in their ceremonial life. They held the belief that the well-being of their herds and flocks depended upon healthy conditions maintained in the community, and that any break in their traditions and customs was bound to affect their stock adversely.

In their ritual meals cows and ewes were used, and on certain occasions such animals were sacrificed to the tribal deities directly. At the great annual rain-making ceremony of the Naman, pregnant cows and ewes were killed to typify fertility, their uteri being preserved till after the feast when the uterine fluid was poured through a fire close to a river bank into the river below. At the same time milk and fat were thrown into the fire. When suffering a long period of drought a similar ceremony was performed in which a pregnant heifer was slaughtered. After a Hottentot's funeral his widow had to scatter the contents of an animal's stomach over the cattle kraals, saying, "let there be plenty of milk", and a similar custom was observed at a girl's puberty ceremony. At a widower's marriage ceremony the bridegroom, after his seclusion, had to sprinkle the cattle and sheep with water before he was again allowed to go among them as usual. Menstruating women must abstain from milking, and girls passing through the puberty rites were led around the kraal where they had to touch the bulls and rams to confer potency upon them. The animals were included in all purification ceremonies, and great care was taken to keep all pollution from their kraals (Schapera, 1930).

As soon as an animal died of disease, the Hottentots immediately moved their encampments to another place. Sparrman (1789) considers that this habit originated almost entirely in prejudice. But it is evident that experience must have taught the Hottentots that staying on in a place where an animal had died of disease, frequently resulted in an additional number of animals succumbing either from poisonous plants or infection. Sparrman himself admits that this "prejudice" "is perhaps one of the principal causes that the cattle of the Hottentots in some measure keep up to their original standard, whilst, on the other hand, those of the Europeans degenerate to a smaller race".

Kolben (1719) states that the diseases of the Hottentots' domestic animals were the same as those occurring among European-owned stock in South Africa, since the latter originate from Hottentot stock and live in the same environment, grazing on similar pastures. The Hottentots were rather reluctant to discuss their animals' diseases with Europeans. "Nothing is known in this country," Kolben writes, "of sheep dying *en masse* as in Europe. Nor do their flocks become mangy or start to cough, or contract other diseases. This is due to the healthy pastures and mild climate under which sheep do not even require salt licks, without which they would not thrive in Europe."

This last statement is of course erroneous, Hottentot sheep requiring just as much salt as those of other countries. Only it was unnecessary to give them special salt rations since there are plenty of natural licks scattered over the arid spaces of Hottentot land.

The phosphate deficiency of South African soils and pastures made itself felt as much among the domestic animals of the Hottentots in the early days as among the European- and Native-owned herds of South Africa at the present time. We have already quoted Sparrman's statement that the Native cattle in the Zuure-velden were given to chew unwholesome substances, such as thongs of leather, chalk and bones, and in default of anything else of the kind, even to gnaw one another's horns.

The Hottentots had among them certain individuals who were looked upon as specialists in the treatment of animal diseases. As soon as it was noticed that an animal was ill, one of these experts was called and asked for his advice. He usually bled the animal first, making incisions with a knife or sharp bone, and then forced some medicine down its throat, decocted from the roots and bulbs of many divers plants. When an animal was unable to pass urine, it was given a kind of earth wax dissolved in water. If a cow's or ewe's udder became inflamed, the swollen parts were treated with an ointment made of tail fat and buchu, a sweet-smelling powder ground by the women from various kinds of aromatic shrub and spores of fungi. Schultze (1907) mentions the following ingredients of buchu:—

(1) The short fleshy leaves of certain species of Mesembryanthemum, or in their stead other herbs or subshrubs, such as slender tuberous roots of a Cyperus species;

(2) various lichens, especially *Parmelia hottentotta* Thbg., which were scraped off rocks;
(3) the dust-like spores of various fungi.

These ingredients were dried, roasted and powdered, and sometimes blended with very fine quartz dust, before being added to the tail fat.

In the event of a flock being seized with an outbreak of disease, an old Hottentot, expert in curing animals, slaughtered three healthy sheep as a sacrifice, one each on three successive days. The meat was consumed by the old men of the tribe, whilst the young men were given the blood and entrails, and the women a broth prepared from the meat. After the meal each of the three groups spent the day and following night apart in singing and dancing to atone for their offences against their supreme deity. If the disease ceased after a time they were filled with joy over the success of their prayers, but if the illness spread further, they laid the blame upon either the sacrificer of the sacrifice for not being good enough. Another old man was selected and the ceremony repeated with three fatter sheep. At the same time the settlement was moved elsewhere.

At certain times the sheep were driven through a smoking heap of wood. On the day determined on, the women milked the cows at dawn and brought the milk to the menfolk who had to drink it all. But the women themselves were strictly forbidden to drink or spill even a drop of it; otherwise the ceremony could not be performed that day. When all the milk had been consumed, some of the men fetched the sheep from the kraal, whilst others prepared a huge fire in an open space, thickly covered it with green branches so as to produce a dense cloud of smoke. When the sheep were brought up, the men ranged themselves in two rows along both sides of the fire leaving a passage between them through which the sheep were driven towards the smoking heap over which they had to pass.

The Hottentots believed that predatory animals, especially wild dogs, would not attack their flocks as long as the smell of the smoke clung to the sheep (Schapera,1930).

It does not seem that the Hottentots had any considerable knowledge of the treatment of stock diseases. Nor did they care very much when an animal died in spite of bleeding and medicines. For they did not bury its carcase nor throw it to the dogs, but consumed it themselves, the owner inviting his neighbours to the feast. Kolben (1719) remarks that the Hottentots' manner of disposing of their dead stock was soon imparted by them to the European settlers at the Cape, " in particular the Governor, Simon van der Stel, who used to treat his male and female slaves to such delicacies, although this brought him but small profit''.

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